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The Edward Stirling Lectures.[1]

LECTURE I.

MODERN TRENDS IN THE SURGERY OF PEPTIC ULCER.

By KENNETH W. STARR,
Sydney.

I DEEM it a great privilege to join with you in the commemoration of Sir Edward Stirling—one of the most gifted sons of South Australia's distinguished traditions.

In these days, when culture takes second place to expediency, it is most invigorating to breathe the purer air of Olympian learning with which his activities and capacity were associated. When he died in 1919, at the age of seventy-one years, he had been honoured by the Realm, by universities, by societies—medical and scientific—by the Fourth Estate, and by the community at large. He was at once a distinguished citizen, a university professor of physiology and an eminent member of his profession, having been president of the British Medical Association in 1888 and of the Australasian Medical Congress in 1905. His outstanding work on surgical subjects, as Dr. Verco reminds us, shows a breadth of outlook, a scholarly depth of thought and an impressive vigour and purity of language which is the reflection of his character and intellect.

His contributions to science, natural history, ethnology, political economy and palæontology stamp him as an *artium magister* and, that unique individual in our profession today, a philosopher and classicist. I have no doubt that he would enjoy as an intellectual exercise the opportunity to enlighten and explore with us the dark recesses of the subject which I propose to introduce this evening, that is, "Modern Trends in the Surgical Management of Peptic Ulcer".

Shortly after his death the text-books began to reflect world teaching on this subject, and it may be meet to examine the surgical recommendations of that period in the light of our modern knowledge.

We were then taught that surgery was indicated in peptic ulcer in the event of perforation, hæmorrhage, cicatrization with obstruction, the fear of malignant supervention and, finally, when efficient medical therapy had failed to cure the condition from the clinical and social viewpoints.

Perforation.

The conservative management of leaking peptic ulcer is not new. When the omentum has blocked the peritoneal stoma, further leakage of contents is as efficiently prevented by natural means as by the sutures of the surgeon, and in many of these cases, recognized and unrecognized, patients have recovered with masterly inactivity.

Recently, however, in view of chemotherapy, ruptured ulcers, with gross leakage into the peritoneal cavity, have been regarded as cases for conservative management. With this latter course I do not agree, having seen two patients die who in my opinion should have been given the benefit of surgery. We know that the recovery rate of ruptured peptic ulcer in competent hands is very high unless the ulcer is associated with gross pyloric stenosis or is carcinomatous; but I do feel that no danger subtends the conservative management of an ulcer which has perforated and in which the following conditions exist: (i) The patient is in hospital. (ii) Adequate doses of penicillin are available. (iii) The stomach is kept empty by continuous gastric suction. (iv) The pain, tenderness and rigidity are confined to the right hypochondrium or,

[1] Delivered on May 31 and June 2, 1949, at Adelaide.

at the most, the right watershed. In effect, tenderness in the left watershed or persistent pain and increasing rigidity demand operation.

Hæmorrhage.

Most patients with hæmorrhage from peptic ulcer recover with conservative management and the number who require surgical intervention is about 3% in my experience in Sydney. The difficulty is to decide, of course, which patients may require surgery (and in that event to have a surgical consultation) and which certainly require surgery as an urgent method of treatment. In general, it may be said that conservative management will be successful in the majority of cases in which the ulcer is recent, the hæmorrhage is small whether single or recurrent, and the general condition does not require, or is easily restored by, blood transfusion. In the last-mentioned circumstances transfusion for gastro-duodenal hæmorrhage should be small and repeated (one to two pints). Large continuous transfusions usually indicate that surgery will be required early and should be employed as a preoperative measure in resuscitation.

Surgery may be required if the hæmorrhage is repeated, but several days intervene between episodes. In the vast majority of such cases the patients recover. Surgery is certainly required in penetrating ulcers with profuse hæmorrhage, and the results of surgery are most successful in those cases in which the ulcer can be found, the bleeding point controlled, and a decision made as to what further surgery is justified at the time of operation.

If a patient with hæmatemesis is known to have a penetrating ulcer, or has suffered for a time with pain in the back, operative management is successful in direct relation to the time at which it is undertaken. Early operation means a high recovery rate. Delayed operation means a very high mortality. In most cases, a surgeon accustomed to gastric resection will perform gastrectomy without serious harm to the patient, but the occasional gastrectomist may well be advised merely to control the bleeding point.

Pyloro-Duodenal Obstruction.

It is not now true that all patients with pyloro-duodenal obstruction should be submitted to early surgery, nor is it true that all such patients should have a gastro-enterostomy performed. It is important to distinguish between complete and incomplete obstruction. Incomplete obstruction may be either acute or chronic.

In the former case the obstruction is associated with œdema surrounding an acute ulcer; the pain and tenderness are also marked. Occult blood is present in the fæces; a test meal shows a high acid curve as a rule, and the obstructive features are indicated by the quantity of vomitus, rarely bile-stained, and the demonstration of gastric residue in a barium meal examination. Such patients respond well to continuous feeding and alkalinization, when an indwelling gastric tube is used. Parenteral therapy with amino-acids, or even human serum, may be necessary if insufficient nutriment is passing through the pylorus.

The management of chronic incomplete obstruction is not different today from what it was twenty years ago, and our fathers probably performed this operation with greater dexterity and competence than we can. Attention, however, should be drawn to the fact that many patients carry on for many years with daily gastric lavage performed by themselves, and that, in incomplete obstruction, operation is not essential to the continuance of life, although it may be to a comfortable existence. Such patients frequently show a considerable degree of gastritis and the acid level may be correspondingly low, but after the performance of gastro-enterostomy and the abatement of gastric inflammation the acid curve may reach its true height and then the Damoclean sword of stomal ulcer hangs over the head of the patient.

In complete pyloro-duodenal obstruction the picture is far otherwise. Surgery in the form of gastro-enterostomy almost invariably produces a fatal outcome. These patients have large gastric residues totalling more than 100 ounces in a day. Tetany may be present; alkalosis with hypochloræmia and azotæmia is almost invariable. When the blood urea is high and gastric retention severe, conservative management by indwelling gastric tube and parenteral feeding with amino acids and normal saline solution is to be commended. The chloride requirements of these patients may equal the chloride content of more than 10 litres of saline solution. If no progress is being made and the gastric residue is still more than 100 ounces *per diem*, jejunostomy is indicated until hypochloræmia and azotæmia have abated and the patient is well enough to withstand the operation of gastro-enterostomy.

Fear of Malignant Disease.

The incidence of malignant disease in gastric ulcer is variously stated as 5% to 20%. Those ulcers mimicking neoplasia are large penetrating ulcers usually situated (*a*) near the pylorus (where they penetrate the pancreas or liver); (*b*) on the lesser curvature extending on to the posterior surface (the so-called saddle ulcer penetrating the pancreas or liver); and (*c*) the juxta-œsophageal or cardiac ulcer penetrating the crus of the diaphragm. In the majority of cases surgery will deal effectively with the pyloric and lesser curvature lesions, but the latter ulcer is indeed a problem. Surgical resection is difficult because total gastrectomy is required and the mortality and morbidity resulting from this procedure are well known.

Many of these large ulcers are associated with azotæmic conditions resulting from genito-urinary or hypertensive disease, and with improvement in the latter and reduction in the azotæmia, the ulcer not unusually heals.

In these cases it is important to examine the ulcer with X rays at intervals of three to four weeks to determine whether it is healing, stationary or progressive. Many physicians are aware that these ulcers can and do heal. It is well to treat them surgically if they remain stationary or are progressive. The problem ulcers at the cardiac end are best managed by vagotomy and local resection.

Failure to Respond to Conservative Management.

It has been our custom to teach that trauma from food particles, focal sepsis and arteriolar spasm may severally cause acute peptic ulcer. These lesions once established fail to heal because of hypersecretion and high gastric acidity, hyperperistalsis and failure of the gastric neutralizing mechanisms. The last-mentioned mechanisms are commonly under-estimated, but while the phenomenon of reversed peristalsis is well known to us, the exact nature of the actual layer which covers gastric epithelium is more in doubt. Recently, the existence of a fibro-cellular layer demonstrated by microincineration has led Duran-Jorda (1946) to assume that erosion of this layer is fundamental to the ætiology of peptic ulcer. Others assume that protective mucus is dissipated by mucolytic enzymes secreted in excess as a result of vagal hyperactivity.

The importance of the vagal element in secretion during the earlier phase of digestion is familiar, and the high acid curve during the first one and a half hours of gastric secretion contains a large acid component of vagal origin. After the first hour, however, the hormonal contribution predominates. We do not yet know the significance of the hormones arising from the pyloro-duodeno segments as the result of contact with food, but much work has elucidated the importance of the vagal contribution, that is, psychic juice. We know that this juice is not limited to the phase of gastric digestion, but is continuous day and night and may reach high figures (even 500 millilitres, representing 80 millilitres of acid equivalent). On this account the elimination of the psychic juice has recently been added to our surgical armamentarium by section of the vagus nerve.

The earlier operations were supradiaphragmatic. This route was employed as the result of arguments derived from surgical experience in the sympathetic nervous system. It was feared that regeneration of the nerve would rapidly occur. We now know that these fears are not completely justified. The disadvantages of this approach are that chest complications are not infrequent,

the pathological viscus (the stomach) is not seen or felt, and owing to the complexity of the nerve from the back of the pulmonary hilum to the diaphragmatic zone, complete neurectomy may be difficult. A small segment of residual vagus nerve is sufficient to restore the secretion of psychic juice.

The infradiaphragmatic operation has therefore gained in popularity, being easily performed if the left lobe of the liver is mobilized and retracted by dividing the coronary ligament. When the œsophagus is stretched, the nerves stand out like bowstrings because of their inextensibility. It is important to ensure the division of all vagal branches and to inspect the stomach carefully to satisfy oneself that the lesion is benign and that the stomach is not hypertrophied or dilated, indicating obstruction. In the latter circumstances, gastro-enterostomy is required. Effective vagal section produces achlorhydria or at least marked hypochlorhydria and a paresis of the gastric musculature, necessitating an indwelling intragastric tube for two to three days until the residue diminishes in quantity.

The completeness of vagal section is tested by estimating (*a*) the night fasting juices, and (*b*) the quantity of acid secreted following the state of hypoglycæmic shock produced by the injection of 16 units of insulin.

When gastric obstruction is present to any degree and vagal section produces paresis, gastro-enterostomy should be performed. In my hands, posterior gastro-enterostomy has on two occasions proved ineffectual and the stomach dilatation has become very distressing. I have found better results to follow the performance of the Lahey type of gastro-enterostomy on the greater curvature at its most dependent part.

Gastrectomy.

Vagal section is at the moment on trial. More than half of the thirty-six patients submitted to vagal section at the Concord Repatriation Hospital have since returned with recrudescence of acid secretion and recurrence of symptoms. This does not apply to the same extent to those cases in which vagotomy has been accompanied by gastro-enterostomy, and nine patients in our series maintain low acid levels and reasonable digestive comfort if the symptoms of gastro-intestinal stasis are ignored. Subtotal gastrectomy therefore remains our chief standby for the control of acid in peptic ulcer. It is a serious reflection upon our physiological knowledge that surgical eradication should prove our chief line of attack. Techniques have considerably improved. My mortality rates in the performance of subtotal gastrectomy for uncomplicated peptic ulcer have shown considerable diminution. The aftermath of the operation, however, is not quite so free of worry to both patient and surgeon. Patients with a bad psychoneurotic background enjoy the sequelæ of gastrectomy to a very high degree. The complications and sequelæ are substantial deterrents to the occasional gastrectomist. These complications produce a mortality rate of 2%. Chest complications of varying degree occur in a great majority of the cases, and gastric retention may be troublesome, necessitating use of the indwelling gastric tube for several days. While better technique reduces the complications, even good judgement does not eradicate the sequelæ.

After gastrectomy, the great majority of patients fail to thrive, and stabilize their weight at a figure about one stone below that of their pre-operative condition. This is of little significance in many sedentary occupations, but in one series of 100 patients more than 50% could not return to heavy work, and 20% complained of discomfort with nausea and vomiting for more than a year after the operation.

The so-called "dumping syndrome" is well known and is receiving considerable publicity in the literature. Giddiness, flushing, faintness and nausea usually arise about half an hour after the meal, more particularly the largest meal of the day, and harry the patient's well-being. Experimental research has indicated that this condition arises from jejunal distension following the rapid emptying of the stomach. In many of these cases abnormal sugar curves of the lag type are present, and the hormonal factors have also received study. The report of such sequelæ after the operation of vagotomy, in which there is grossly delayed emptying of the stomach, would seem to indicate that the answer to this problem does not lie totally in the upper jejunal loops and, when taken in conjunction with the patient's failure to thrive and with the lag type of sugar curve, promotes the hormonal ætiology as a possible alternative.

The recent work of Kahlson (1948) in Sweden seems to indicate that the duodenal mucosa has an important digestive function and we do not yet know the effects of pyloroduodenal excision on protein, fat and carbohydrate metabolism. This consideration has prompted me to attempt vagotomy with pyloroplasty as an answer to this difficult problem and the results will be watched with interest in view of the fact that the duodenum is retained.

Further, stomal ulcer may follow subtotal gastrectomy, the incidence being directly proportionate to the amount of residual stomach. Visick (1948), in 600 cases, has reported complete freedom from this unfortunate complication by the application of new principles to the operation of subtotal gastrectomy.

Most surgeons are agreed that it is imperative to remove the pylorus, pyloric antrum and lesser curvature (at least as high as the left gastric artery). Greater latitude has attended the resection of the greater curvature. A "high" gastrectomy was previously a popular operation, the greater curvature being divided immediately above the entry of the left gastro-epiploic artery.

However, the complete liberation of the greater curvature, except at the upper *vas brevis*, permits a measured gastrectomy with less residual stomach than in other methods. Further, this liberation permits more ready suturing owing to the greater freedom of the gastric stump.

The incidence of stomal ulcer after gastrectomy varies between 5% and 10% in the various series published, and the efficacy of vagal section in reduction of symptoms, with resultant healing of the ulcer, is now familiar to us all. It appears to be the most definite and reliable of all the indications for vagal section.

Conclusion.

In retrospect we may recall those days of ardent gastric surgery when gastro-enterostomy was performed as a panacea for all resistant and complicated peptic ulcers. We should not be too zealous in our condemnation of this surgical phase, but remember that many good results accompanied the great number of obvious, and greater number of occult, stomal ulcers whose healing has closed many stomata.

Within a decade of Sir Edward Stirling's passing, the biochemistry of alkalosis, hypochlorræmia and azotæmia incidental to gastric obstruction had made considerable progress and lent a sounder scientific aspect to the assessment of these cases. Within two decades high subtotal gastrectomy had become a perfected technical procedure and gastric aspiration with the concomitant registration of fluid balance had achieved a widespread and rational acceptance. Within three decades vagotomy had engendered a new interest in the physiology of gastric secretion and presented surgeons with a new method of attacking the problem of vagal hypertonicity and its complications.

Progress, however, is slow because of our lack of knowledge of the basic ætiology of peptic ulceration. In this regard we cannot hope to bring the matter to a satisfactory conclusion until we can pronounce ourselves successful in what Burton was pleased to call "chasing the nimble and retiring truth".

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Duran-Jorda, Frederic (1946), "Micro-Incineration Study of the Flat Epithelial Layer Covering the Alimentary Tract", *The British Journal of Surgery*, Volume XXXIII, page 346.

Kahlson, Georg (1948), "Nervous and Humoral Control of Gastric Secretion", *British Medical Journal*, Volume II, page 1091.

Visick, A. Hedley (1948), "Study of the Failures after Gastrectomy", *Annals of the Royal College of Surgeons of England*, Volume III, page 266.

A SERIES OF CASES OF INDIRECT INGUINAL HERNIA TREATED BY THE METHOD OF INVERSION OF THE SAC.[1]

By C. CRAIG, M.D., M.S., F.R.A.C.S.,
Surgeon Superintendent, Launceston General Hospital, Launceston.

THIS paper is a continuation of one published recently,[(1)] in which an attempt was made to show that in at least 80% of cases of hernia a simple operation whereby the sac only was dealt with was sounder than a plastic operation which involved interference with the posterior wall. Two suggestions were made, however, as to methods by which the simple operation could be improved. One was the insertion, in a special way, of a piece of skin or fascia. This special method of insertion has not so far been tried. The other was the method of treating the sac by inversion instead of by twisting, ligation and cutting off. This paper deals with the results of the inversion method.

Two Possible Causes of Recurrence.

Before the method of inversion is discussed, however, it is desired to place on record two observations that may throw light on the causes of recurrence after the simple operation.

The first observation has to do with the method of twisting the sac before ligation, which was introduced to surgery by Ball. Ball's twist was used by me in every case in the preceding series. I stated that I had no doubt that I had removed the whole sac in every case. I now realize that I may not have done so.

A male patient, aged forty-six years, complained of having had a hernia for six months. At operation an ordinary indirect sac was found on the left side and was dealt with by freeing, twisting, ligating and cutting away the excess sac. I felt quite certain that the whole sac was enclosed in the ligature. When the patient was asked to strain, however, a definite localized bulge the size of a marble appeared immediately inferior to the ligature. The ligature was cut, but it was found that the bulge could not be obliterated by further twisting unless undue violence was used.

It is apparent, therefore, that in certain cases there is some anatomical reason which prevents the whole sac from being taken up when the sac is twisted (Figure I).

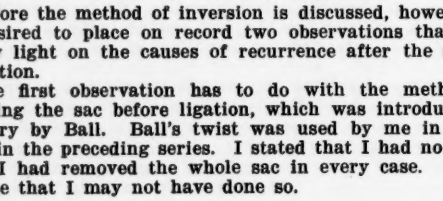

FIGURE I.

Ball's twist was used and the patient was asked to strain. A bulge appeared below the ligature. It was not possible to obliterate this bulge by further twisting. Unless discovered such a bulge might lead to recurrence.

It shows how essential it is that the patient should strain after the sac has been ligated. I have to confess that I have at times not insisted on this being done as well as it should have been. In this regard the form of anæsthesia is important. If spinal anæsthesia is used the patient can rarely get up enough pressure. Even if local anæsthesia is used it is often difficult to induce the patient to strain sufficiently. The best method is to use general anæsthesia and, at the appropriate moment, to allow the anæsthesia to become light so that the patient vomits. The strain produced by vomiting is terrific and all surgeons new to the operation should study the inguinal canal whilst the patient is vomiting, particularly to observe the sphincteric action of the conjoined muscle.

The other observation shows that occasionally another sac is present.

This condition was found in a female patient, aged thirty-five years. A left indirect sac was removed in the usual way. It was then seen that another sac was lying anterior to the cord and projecting about 2·5 centimetres into the canal. This sac, which did not appear ever to have been opened, had the same anatomical relationship to the round ligament that an ordinary sac has, but its mouth was about 2·0 centimetres posterior to that of the ordinary sac (Figure II).

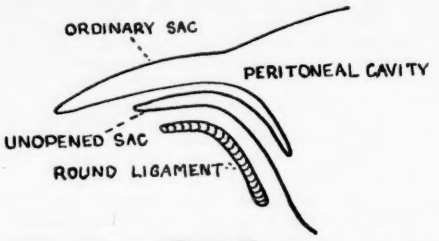

FIGURE II.

After the sac containing the hernia had been removed, another unopened sac was seen lying beneath it. Unless discovered such a sac might lead to recurrence.

It is obvious that the presence of a second sac should always be looked for. It is hardly likely to be missed if the round ligament, vas and pampiniform plexus are separated from the peritoneum well into the pelvis, as should always be done.

These two observations illustrate well that the basic procedure in every operation for hernia is the obliteration of the sac.

Technique of the Inversion Method.

The technique of the inversion method was described in the previous article. Two illustrations from the article demonstrate the two methods that may be used. One is inversion into the abdominal cavity (Figure III) and the other is inversion by bringing the sac through the abdominal wall (Figure IV). The method that is chosen will depend on the type of sac that is present. This may be summarized in the following way.

Thin, pliable sacs are best treated by inversion through the abdominal wall. As most hernial sacs are of this nature it follows that this will be the method adopted in the great majority of cases.

Sliding herniæ can be treated only by inversion into the abdominal cavity, unless, as is often the case, the sliding hernia can be reduced by dissection.

Thick, heavy sacs should not be brought through the abdominal muscles. However, if it is convenient in the first place to bring such a sac through the muscles, the opening in the muscles should be widened, the sac sewn securely to the parietal peritoneum, and then the excess of sac either cut away or brought up between the peritoneum and the *transversus abdominis*. This having been done, the gap in the muscles is closed. Such thick, heavy sacs, however, are equally easily treated by inversion into the abdominal cavity.

There is one point in the technique that is worthy of notice. In 58 cases, with the idea of producing localized fibrosis, carbolic acid dissolved in olive oil was introduced into the area where previously the neck of the sac lay. At first the method used was to introduce about one or two millilitres into the space with a syringe, but lately a small piece of oxidized cellulose has been soaked in the carbolic solution and placed against the mouth of the inverted sac. There is no evidence whatsoever to show that this method was effective or otherwise.

[1] Read at a meeting of the Section of Surgery, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

The particulars of the clinical material are as follows.

The total number was 130. The patients fell into the following age groups: one to ten years, 14; ten to twenty years, 10; twenty to thirty years, 15; thirty to forty years, 20; forty to fifty years, 24; fifty to sixty years, 22; sixty to seventy years, 19; over seventy years, 6.

Male patients numbered 122 and female patients 8.

One hundred and sixteen of the patients followed active occupations and 14 sedentary occupations.

The duration of symptoms before operation was as follows: up to six months, 61 cases; six to twelve months, 23; one to two years, 10; two to three years, 5; three to four years, 6; four to five years, 4; five to ten years, 17; over ten years, 4.

The number of fingers admitted through the neck of the sac was as follows: half a finger, 5 cases; one finger, 98; one and a half fingers, 12; two fingers, 10; two and a half fingers, 2; three fingers, 3.

High spinal anæsthesia was used in 113 cases, local anæsthesia in 3, and general anæsthesia in 14.

All patients were kept in bed for fourteen days and were advised not to return to work for two months.

The Muscle Sphincter.

One of the great services done by Hamilton Russell to the cause of hernia treatment was to emphasize the importance of the muscle sphincter arching over the cord. It is probably impossible to over-exaggerate the importance of this arch in supporting the underlying fascia. Most of the patients operated on for hernia in this hospital are active workers and there is little need to worry about their muscles. Nevertheless they are instructed before operation to keep their muscles in order by practising a sudden stiffening movement of the abdominal muscles, and this is continued after operation.

There sometimes appear reports of series of cases in which the surgeon has used an extensive plastic operation because he is dealing with a group of patients whose muscles are in bad order. Surely this is indefensible. If there is one thing that the physiotherapists have taught us it is that if there is a muscle present and if its nerve supply is intact, it can be restored to full activity even if it is in a state of atrophy. Obviously the thing to do when atrophied muscles are suspected is to make sure that they have been restored to health before operation.

It is quite possible that the slogan of the future may be: "Restore the posterior wall by physiological means before operation and then deal with the sac in such a way that the fascia is left streamlined."

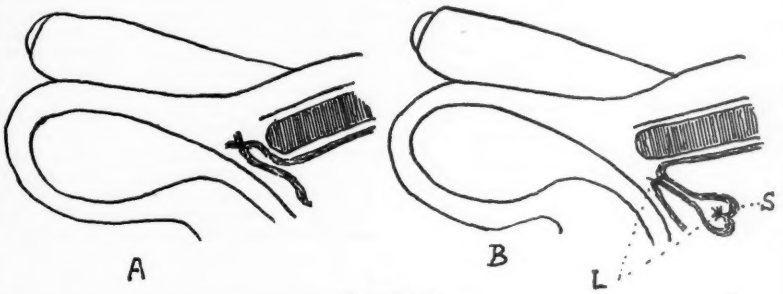

FIGURE III.

From *The Australian and New Zealand Journal of Surgery.*[1] Showing the method of inversion used for sliding herniæ. *A* is before inversion. The sac has been opened for inspection and then closed with a ligature so that no raw surface presents to the peritoneum. *B* is after inversion. The mouth of the inverted sac is closed with interrupted sutures. A purse-string suture should not be used. *S* = inverted sac, *L* = ligatures.

The Results of the Inversion Method.

The inversion method was first introduced on October 9, 1945. Between that date and January 31, 1948, which was six months ago, the inversion method has been used on 130 occasions. During this period only six cases were excluded on the grounds that the hernia was too large. In the early days the method of inversion into the abdominal cavity was used, and 16 patients were treated by this means. Since then the method of inverting the sac through the abdominal wall has been used and this was adopted in 114 cases. There has been a close personal follow-up.

So far there have been three recurrences. The first recurrence is particularly worthy of note.

Hardly was the patient, a man of fifty-two years, out of bed before a bulge was noticeable at the external ring, and it soon became obvious that a direct hernia was present. This was confirmed when operation was undertaken six months after the first procedure. The important point is that, as is usually done, the region of the direct hernia had been explored with the forefinger during the first operation and no sac was found. Now the main idea of my first article was to show that if the simple operation is performed the patient who suffers from indirect hernia is no more liable to direct hernia than if he had never had an indirect hernia at all. In this case, however, the direct hernia appeared so soon after the first operation that it could justifiably be called a recurrence. I was at a complete loss to account for this when I suddenly recollected that it was in this case that I had given to the resident medical officer a demonstration of the strength of the posterior wall. First I had put my finger in and felt. Then she had put in hers. I was not satisfied and again put in mine, and so on, till we had each felt several times. Altogether the posterior wall got a severe handling, and I have little doubt that the direct hernia resulted from this.

This again illustrates what was said in the previous paper, that, although the posterior wall is very strong, it is very delicately set. Since his first operation for the direct recurrence this patient has suffered two further small recurrences—one at each end of the canal. This patient's recurrence was no doubt also contributed to by the fact that there was a "high" insertion of the conjoined muscle—that is, it appeared that it was inserted in such a way that a considerable area of the posterior wall was left uncovered by muscle. As the series has progressed, a note has been made in 91 cases as to the apparent method of insertion of the conjoined muscle in each case. This has been classified as "normal" when the ordinary method of insertion obtained, as "high" when the conjoined muscle joined the rectus sheath at least 2·5 centimetres above the pubic crest and as "moderately high" when the insertion lay between these two. In all, 76 insertions were classified as being "normal", nine as being "high", and six as being "moderately high". It must be made clear that judgement in this matter was formed from appearances in the field of operation. No attempt was made to dissect out the actual insertion. It must also be pointed out that the term "conjoined muscle" has been used rather than "conjoined tendon" to indicate the arch of muscle fibres that bridges the canal. A special note was made as to whether, in the field of operation exposed, this ever appeared tendinous, and in none of the 130 cases did it do so.

The second recurrence was in a male patient, aged fifty-three years, a mechanic by trade. He had had his hernia for two months before his operation in January, 1946. A thin, pliable sac admitting one finger was present, and as this was in the early days of the inversion method, this was inverted not through the abdominal wall, but into the abdominal cavity. I would not now consider this a suitable method for this type of sac. The recurrence appears to be indirect, but so far it has not been treated surgically. The recurrence appeared two and a half years after the operation.

The third recurrence was in a male patient, aged sixty-seven years, a farm hand. He had had his hernia for six years before operation in October, 1945. At operation it was found that the sac was large. Although normal in

position, the conjoined muscle was poorly developed. Again the sac was treated, not as I would treat it now by inversion through the muscle, but by inversion directly into the abdominal cavity. As two of the three recurrences have taken place after the use of this technique, it would seem that it is not suitable for any type of sac except very thick sacs, or when a sliding hernia is present. The recurrence appeared two years and eight months after the operation and appears to be indirect.

So far, then, the recurrence rate is 2·3%; but it cannot be too strongly emphasized that it is too soon to assess the correct recurrence rate. Some time will have to pass.

It is desired now to direct particular attention to recurrence through that part of the abdominal wall through which the inverted sac has been brought. There have been no such recurrences in this series; but in three other cases—two of direct "tubular" hernia and one of femoral hernia—there have been recurrences in this region. In each case the original sac was a thick, heavy one, and it is obvious that this method is unsuitable for such sacs. In these cases either the sac should be inverted into the abdominal cavity or, if it is brought through the muscles, it should be securely sewn to the parietal peritoneum, the excess cut away or else drawn up between the peritoneum and the transversus, and the slit in the muscles closed. If the excess is cut away, care must be taken that the bladder is not opened. The possibility of injury to the bladder is always present in any hernia operation, and attention will now be directed to this possibility in the method of inversion through the muscles.

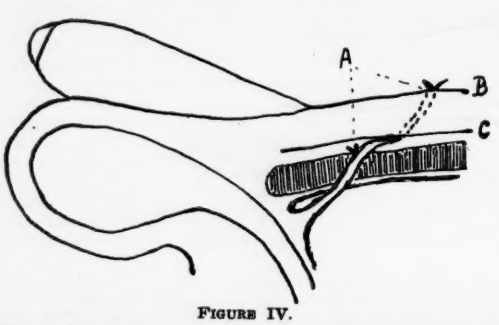

FIGURE IV.

From *The Australian and New Zealand Journal of Surgery.*[1] Showing the method of inversion for the ordinary type of sac. Two sutures hold the sac to the internal oblique and one suture from the end of the sac is passed through the skin and tied. The sac may be gently twisted after inversion. *A* = ligatures, *B* = skin, *C* = external oblique.

Damage to the Bladder.

In two cases in which inversion through the abdominal muscles had been performed, a series of events took place which raised the possibility of damage to the bladder. In each case, in the few days after the operation, the temperature and pulse rate rose. An area of discoloration appeared which surrounded the wound and extended for several inches up onto the abdominal wall. In each case the patient suffered from paralytic ileus and was very ill. It is thought that one of the stitches which were placed to hold the sac to the abdominal wall had penetrated a diverticulum from the bladder and that leakage had resulted. It is well known that a diverticulum of the bladder or even the bladder itself may appear in the inguinal canal alongside the sac. In the usual method of dealing with the sac, however, such a prolongation is safely tied in the ligature that is placed round the neck of the sac. When inversion through the abdominal wall is carried out, it should be remembered that a diverticulum of the bladder would appear on the outer side of the inverted sac, and steps should be taken to avoid it.

The Possibility of Intestinal Obstruction.

One of the disadvantages of this operation is the fact that the peritoneum has to be opened. When such an event takes place there is always the possibility of intestinal obstruction from adhesions. As it has happened, two of these patients suffered from intestinal obstruction within two days of the operation. In both cases, however, the obstruction was due not to the recent operation, but to previous operations performed for appendicitis. However, the fact that the abdomens had to be opened gave me an opportunity of inspecting the inverted sacs. In neither case was there any sign of the presence of adhesions. Moreover, in neither case was there any pocket or canal into which the bowel could penetrate. One of these patients died as the result of the intestinal obstruction.

Conclusion.

It is too soon for final conclusions to be drawn from this series. Another two years at least will have to pass. Nevertheless, it must be pointed out that in 50 cases two years have elapsed since the operation and in another 45 cases one year has elapsed. The results, therefore, must be regarded as satisfactory so far. My own impression is that, so long as only the usual thin pliable sacs are brought through the abdominal wall, the method is a good one and apparently is a little more certain than that when Ball's twist is used. The chief difference between the two procedures is that when the ligature and excision of the sac method is used it is impossible to avoid leaving a projection of peritoneum in the region of the internal ring. When the inversion method is used, however, there is no such projection.

I should like to emphasize that again a series of patients has been satisfactorily treated by a method which does not involve interference with the posterior wall. During this same period 35 patients were treated by the modified Hamilton Russell method that was described in the previous paper. None of these have had a recurrence. Therefore, in all, 165 patients have been treated by a simple method with a recurrence rate of 1·8%.

Moreover, whereas in the series described previously 30 patients were excluded because the herniæ were too big, in this series only six patients were excluded, so that, of the total number presenting for operation, 96% were considered suitable for operation by a simple method.

Summary.

1. Two observations which may throw light on recurrence of hernia after a simple operation are recorded.

2. A series of 130 cases of indirect inguinal hernia, treated by the method of inversion of the sac only, is presented. This series was commenced nearly three years before presentation and was concluded six months before presentation.

3. There have been three recurrences, a recurrence rate of 2·3%. It is emphasized that more time will have to pass before a final assessment can be made.

4. It is pointed out that of the two main methods of producing inversion the method of inversion through the abdominal wall is the only suitable one for the ordinary thin, pliable sac. One hundred and fourteen patients were operated on by this method. There was only one recurrence—a direct recurrence which was thought to be the result of trauma at the operation. Sixteen patients were operated on by inversion of the sac into the abdominal cavity. There were two recurrences in this group, both apparently indirect. As the sac in both cases was thin and pliable, it is considered that the wrong technique was followed in these two cases.

5. In 58 cases carbolic acid in olive oil was introduced into the area occupied by the neck of the sac, with the idea of producing fibrosis. There is no evidence to show whether this was effective or not.

6. During the same period 35 patients were treated by the method of twisting, ligating and shifting the neck of the sac. There were no recurrences in this series, so that in all 165 patients have been treated by a simple method not involving interference with the posterior wall, with a recurrence rate of 1·8%.

7. During this period only six cases were excluded on the score of size of the sac, so that 96% of all patients presenting were considered suitable for the simple operation.

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CLINICAL USES OF THE BASAL BODY TEMPERATURE GRAPHS IN PRACTICE.

By ALAN GRANT,

From the Sterility Clinic, The Women's Hospital, Crown Street, Sydney.

IT is now becoming more commonly known that there is a biphasic variation in the basal temperature of normal women between one menstrual period and the next, so that the level of the daily basal temperature is higher after the occurrence of ovulation than it was before. In order to discover this the temperature must be taken after the patient has been at rest.(1)

Not so well appreciated, however, are the valuable and practical applications of this temperature variation, and it is the purpose of this paper to translate the implications of the biphasic temperature curve into terms of diagnostic action and clinical utility. The conclusions are based on a study of cycles taken by patients, mostly at the sterility clinic of The Women's Hospital, Crown Street, Sydney (582 patients recorded on graphs 1509 cycles).

One can use the basal body temperature graph (hereinafter referred to as the B.B.T. graph) as a diagnostic instrument only if one is familiar with the normal graph, and therefore we must halt here for a description of what the B.B.T. graph is like both in the non-pregnant normal female and in the pregnant patient.

The Normal Curve in the Non-Pregnant State.

1. The normal curve in the non-pregnant state is biphasic with a higher plateau of temperature in the second part of the cycle.

2. This rise in temperature is usually preceded by a drop which corresponds to the probable date of ovulation. This drop will not be observed in all temperature charts, but it is probably present in all cycles if the temperature is taken close to the actual time of ovulation.

3. The height of the rise is two-fifths of a degree or higher and must be maintained at that elevation to be a correct indication of the luteal phase.

4. The length of the luteal phase has been categorically stated to be fourteen days, but according to our observations it is not.(3)(4)(5) In our series 90% of patients ovulated between the twelfth and sixteenth days before the onset of the next menstrual period. In the remaining 10% the luteal phase varied from ten days to eighteen days consistently. In this anomalous group the luteal phase was more often of shorter duration than fourteen days than it was the opposite. It will be noted therefore that 90% of patients have a luteal phase of about fourteen days.

5. The temperature level of the luteal phase frequently drops the day before the period is due.

6. Double ovulation may occur. Patients will occasionally but not consistently return a graph which shows two temperature rises, one in the first half of the cycle and one in the second half or last fourteen days. This type of chart has been considered by some(2) to represent double ovulation, but so far the assumption has not been proved. Such a chart is illustrated in our series. We are still investigating the suggestion that such patients may be producing two ova at separate time intervals during the one intermenstrual cycle. In these cases other well-known causes for elevation of temperature have been eliminated, such as alcohol, coryza and migraine. (Note: In the specimen graphs exhibited it will be noted that day number one of the cycle is taken to be the first day after the menstrual period has ended. This makes for easier handling and sorting of a mass of graphs. In most text-books the first day of the cycle is assumed to be the first day of the menstrual period.)

The Normal Curve in Pregnancy.

1. The curve during the cycle preceding the first missed menstrual period is similar to that associated with the non-pregnant state.

2. However, the day before the period is due the temperature fails to drop.

3. The menstrual period is then missed.

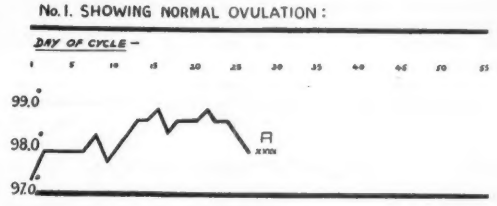

FIGURE I.
Normal ovulation; A = menstrual period.

4. Subsequent to the first missed period, the level of the temperature in the luteal plateau usually rises to 99° F., or higher, and is maintained there for several months, after which it commences to drop to the level it occupied after the last period. For a diagnosis of pregnancy it is necessary only for the temperature to remain at the same level after the amenorrhœa as before it occurred, though a level of 99° F., or over, is more usual than any temperature level below it.

5. Death of the ovum is followed by a fall in temperature.

The Clinical Uses of the B.B.T. Graph.

Occurrence of Ovulation.

One of the commonest indications for recording a B.B.T. graph is to determine the presence or absence of ovulation for any reason, for example, (i) to check X-ray therapy or other treatments used for amenorrhœa of endocrine origin,(6) (ii) at the menopause, to check whether the patient can become pregnant, and (iii) in sterility studies.

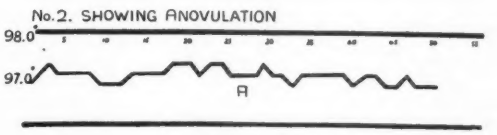

FIGURE II.
Anovulation; A = menstrual period due.

In the treatment of functional amenorrhœa by hormones, gonadotrophic substances being used or substitutive programmes like those proposed by Kaufman or Zondek or thyreoid extract, it is desirable to check the presence or absence of ovulation after menstruation has occurred. This is referred to by American authors as looking for the "fertility salvage" in any particular case. Some would refer to it as determining whether the patient is exhibiting true menstruation or pseudomenstruation.

At the time of the menopause some patients desire to know whether they can dispense safely with contraceptives. The B.B.T. graph offers a method of deciding whether the cycles are all anovulatory (and pregnancy-proof) or not so.

It is in regular studies of the childless couple that the B.B.T. graph finds its greatest usefulness, as one can be tolerably sure whether ovulation is occurring or not. Too many doctors prescribe some preparation such as

vitamin *E* without knowing what are the causes of the childless marriage in that particular married couple. Vitamin *E* seems to be endowed with perennial youth. No sooner is it found to be of doubtful value for one group of complaints, such as female sterility, or for spermatogenesis, than it reappears on the therapeutic stage cast in another role. In my opinion it is time that we ceased referring to it as the "fertility vitamin". It prevents death of the fœtus in female rats which have been previously deprived of it. In the field of sterility nothing further has been proved, but much more has been asserted.

Time of Ovulation.

Another use for the B.B.T. graph is to determine the day in the cycle when ovulation occurs in any particular patient as an individual. This method has been correlated with endometrial biopsy and found to be as accurate. This work was carried out in 80 cases by the Wyeth Fellow

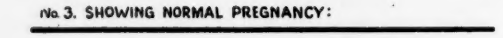

FIGURE III.

Normal pregnancy; A = menstrual period due.

in Obstetrics, Dr. Ian Swain, by courtesy of Professor Bruce Mayes. The actual day of ovulation is of use in the following clinical problems: (i) to find the best time for intercourse in patients suffering from sterility; (ii) to work out the "rhythm method" of contraception; (iii) to work out artificial insemination timing.[7]

Diagnosis of Pregnancy.

If a previously regular patient misses a menstrual period and the luteal plateau of elevated temperature is maintained for fourteen days after the onset of amenorrhœa the patient is almost certainly pregnant. Applied in this manner the test is as accurate as the Aschheim-Zondek pregnancy test. Particular note should be made of the word "if" and the phrase "maintained for fourteen days after the onset of amenorrhœa". It will be appreciated that the B.B.T. test for pregnancy therefore effects a saving in mice, money and manpower.

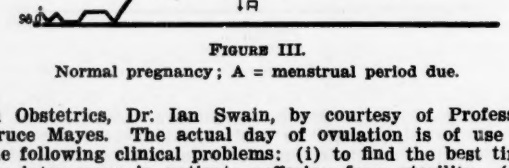

FIGURE IV.

Threatened abortion and death of ovum; A = ovulation; B = menses due; C = positive response to Aschheim-Zondek test; D = fall in temperature, no cause being found; E and F = light hæmorrhage; G = heavy hæmorrhage, curettage.

Abortion, Threatened and Missed.

The elevated level of the luteal phase is maintained into pregnancy. If the ovum dies, the temperature level falls to the level it was at immediately after the previous and last period. Thus we may know (i) whether a "clean" abortion is inevitable, and (ii) whether a "missed" abortion has occurred.

Ectopic Pregnancy.

The position of the B.B.T. chart in relation to ectopic pregnancy is similar to that of the Aschheim-Zondek test in so far as, if enough villi are alive and actively producing hormone, then the evidence of both the Aschheim-Zondek test and the B.B.T. chart will be positive for pregnancy. Therefore if the evidence of the B.B.T. chart is positive for pregnancy and a painful tumour develops in one fornix, the diagnosis is "probably an active tubal pregnancy". If the evidence of the B.B.T. chart is not positive for pregnancy and a painful tumour develops in one fornix, then the diagnosis may still be "a dead or dying tubal pregnancy". A negative result to the test is therefore not exclusive of ectopic pregnancy, any more than the occurrence of a negative result to the Aschheim-Zondek test is exclusive of it.

Dysmenorrhœa.

"The only fact known about functional dysmenorrhœa is that ovulation is essential to cause it", according to Hamblen.[8] Most other authors agree with this statement. Dysmenorrhœa may therefore be treated by suppressing ovulation with adequate doses of some natural or synthetic œstrogen. The B.B.T. chart may be used to

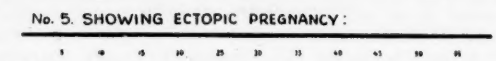

FIGURE V.

Ectopic pregnancy; A = red blood, not normal menstrual period; B = slight hæmorrhage; C = rupture, operation.

check whether the dose of œstrogen has been adequate to suppress ovulation. F. Ingersoll uses the "œstrogen test" before deciding whether the patient is suitable for the treatment of dysmenorrhœa by the operation of presacral neurectomy.[9] If œstrogens cure the dysmenorrhœa then presacral neurectomy will also cure it. If œstrogens fail to cure it, he concludes that there is a large psychological element and avoids operation.

An Aid in the Surgery of Sterility.

Mrs. M. had previously had one ovary and one Fallopian tube removed. The results of all other investigations on husband and wife were normal. The remaining tube was patent, but the only remaining ovary presented as an ovarian cyst four inches by four inches in diameter. A B.B.T. graph revealed that, nevertheless, the cystic ovary was ovulating. At operation therefore a tedious dissection was undertaken to remove the cyst and leave the small ledge of ovarian tissue behind, because the B.B.T. graph had indicated that it was functional. The cyst was reported on as follicular in origin. This patient now has regular menstruation and an opportunity of becoming pregnant, as the B.B.T. graph taken after operation also indicated the salvage of ovulation.

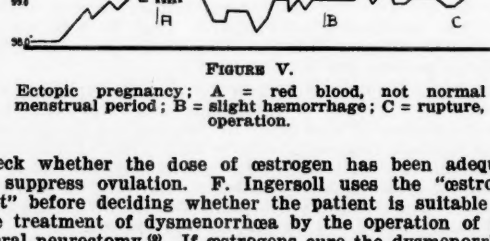

FIGURE VI.

Double temperature rise in one cycle; A = menstrual period.

Final Remarks.

When to Take the Temperature.

The temperature is best taken in the morning before rising, drinking or smoking. The thermometer should be left *in situ* for two to three minutes.

Where to Take the Temperature.

The best situation for taking the temperature in my opinion is the rectum. The disadvantage of the vagina is the fact that thermometers are occasionally lost. The disadvantages of the oral route are as follows: (i) some people breathe through their mouth all night; (ii) others fit a denture immediately on waking, which alters the mouth temperature; (iii) in practice temperature graphs

recorded from the mouth are "spiky" and erratic, whereas those recorded from the rectum are smooth and easy. In other words a temperature chart which has been recorded from rectal readings is more graphic and easier to interpret.

False Temperature Rises.

Occasionally one or two temperature readings may be elevated owing to extraneous causes, such as coryza or migraine or an alcoholic adventure; all these items should be recorded on the chart.

Conclusion.

This test is not purely of academic interest. It can be used as the "poor man's test" for ovulation and for pregnancy. It should be of inestimable benefit to those engaged in general practice, especially for the diagnosis of pregnancy. In this connexion it is worth repeating that "if a previously regular patient misses a menstrual period, and if the luteal plateau of elevated temperature is maintained for fourteen days after the onset of amenorrhœa, then the patient is amost certainly pregnant".

Acknowledgements.

I wish to thank Dr. R. Mackey, and also Dr. I. Swain and Dr. D. McGrath of The Women's Hospital for their help in the sterility clinic and also those members of the honorary staff who have referred cases for investigation. Dr. J. Murray Moyes, pathologist at The Women's Hospital, kindly reported on the endometrial biopsies. Sister Johnson and Sister McGovern and Mrs. McKibbins, the secretary of the clinic, have explained these graphs to the patients with commendable effort and restraint. Finally I wish to thank Professor Bruce Mayes for his advice and enthusiasm and for allowing me to use the services of Dr. Swain and Dr. Crowe, Wyeth Fellows in Obstetrics.

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A REVIEW OF THE USES OF RADIOACTIVE IODINE, WITH A NOTE ON PRELIMINARY WORK IN MELBOURNE.[1]

By R. KAYE SCOTT, M.D., M.S., F.R.A.C.S., F.F.R., D.T.R.E.,

Honorary Radiotherapist, Royal Melbourne Hospital.

IODINE, as chemically known, has an atomic weight of 127 and an atomic number of 53. It is composed of one stable isotope only, but eight other unstable or radioactive isotopes are described. Fifty-three protons are present in each nucleus, but the number of neutrons present varies, and so the combined number of protons and neutrons in the eight radioactive iodine atoms varies between the limits of 124 and 137. The half-lives vary from thirty seconds for $_{53}I^{137}$ to thirteen days for $_{53}I^{130}$.

[1] Read at a meeting of the Section of Radiology and Radiotherapy, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

Fermi (quoted by Leucutia, 1946) was the first to produce radioactive iodine (I^{128}) and very soon several other isotopes were produced in the Berkeley cyclotron by bombardment of tellurium. Active iodine may be produced in the pile, either as a fission product of uranium or by bombardment of tellurium with slow neutrons.

The possibilities of radioactive iodine for the study of thyreoid physiological and pathological processes were quickly realized, and Hertz (1941) initiated the clinical work using I^{128} of twenty-five minutes' half-life. He showed that some radioactive iodine became localized within the thyreoid within ten minutes of its oral administration.

Hertz's work showed that the normal thyreoid picked up iodine very rapidly, while there was little iodine absorption elsewhere; in pathological conditions of thyreotoxicosis the avidity for iodine was greatly increased, and he and his colleagues were able to report two series of cases of the therapeutic use of radioactive iodine in which 20 out of 29 and 20 out of 22 patients were regarded as cured. However, it was very significant that the irradiation of the thyreoid parenchyma did cause cessation of gland function and, with increasing dosage, its destruction. The development of myxœdema of varying degree was constantly observed after the administration of radioactive iodine for these therapeutic purposes (Hertz and Roberts, 1946; Chapman and Evans, 1946).

This work was followed immediately by reports of the use of radioactive iodine for carcinoma of the thyreoid. Hamilton (1942) observed that in his cases of carcinoma of the thyreoid the neoplasm did not pick up iodine, and so he concluded that radioactive iodine could not play a large part in the treatment of this disease. But soon other workers recorded cases of carcinoma of the thyreoid in which some pick-up by the neoplasm was observed (Franz *et alii*, 1944; Leiter *et alii*, 1946).

Tracer Tests.

A very considerable amount of work has now been carried out in the use of I^{131} for tracer tests. The tracer dose varies with different workers. Quimby advises 60 microcuries given orally for adults, or an amount of six microcuries per stone for children (Quimby and McCune, 1947).

Keating used tracer doses of 100 microcuries in his studies of excretion of iodine (Keating *et alii*, 1947).

Larger amounts are used in testing for pick-up capacity of thyreoid carcinoma metastases, and I saw Richard Chamberlain in Philadelphia using an amount of 400 microcuries for this purpose.

The Risks of Tracer and Therapeutic Doses.

Marinelli (1942) has worked out the tissue dosage resulting from the administration of radioactive isotopes, and Hertz and Roberts (1946) expressed their dosage in relation to the thyreoid in terms of *r* units.

The risks of dosage to the thyreoid may be immediate or delayed. But therapeutic effects are not seen with tracer doses. Administration of amounts of four to six millicuries of I^{131} produces doses of well over 1000 *r* in the thyreoid. Large doses may produce minor or major thyreoid crises resulting from destruction of the walls of the acini and rapid absorption of colloid. However, such effects should not be observed with doses of the order of four millicuries as used for thyreotoxicosis. However, slight swelling and tenderness of the gland, tracheitis with cough, or slight dysphagia may be noted in the reactionary stage. These usually pass rapidly, but a state of hypothyreoidism of a degree varying with the dosage is the usual sequela.

Tracer doses are designed to give whole body irradiation within the limits regarded as of "safe tolerance". Under these conditions there is no risk to hæmatopoietic tissues from tracer doses, and even therapeutic doses do not harm the blood-formative tissue, as no localization whatever occurs within the marrow.

Little risk to impairment of reproductive activity of the spermatogonia will follow such dosage, and so no question of production of permanent or temporary sterility will arise. Any effect on the undeveloped gonads of children

might be expected to be of even less degree on account of the lack of development and the consequent decreased sensitivity of resting reproductive tissue.

But grave doubts exist as to the possible production of genetic effects. Such effects might be produced by external radiation just as much as by internal radiation emitted by circulating radioactive isotopes. Many of the American workers have been frankly worried by these possibilities, particularly as so many of the scientific workers are receiving constant dosage, even though it is regarded as within safe tolerance limits. We do not know what constitutes safe tolerance in regard to production of these genetic effects, though we do know that one quantum hit in the right spot is liable to cause a mutation, and the statisticians have been to much trouble to work out what is the risk of damage or the degree of safety. One does not derive much sense of security when one realizes that one millicurie represents 37×10^6 disintegrations per second and that one quantum hit may produce a mutation, the effects of which may not appear for six or eight or more generations.

For this reason I have not felt justified in submitting to even tracer tests patients within the reproductive age limits. It has been said that isotopes should not be used for therapeutic purposes in any patient with a survival chance of over a few years. Much work yet remains to be done before these substances can be used with impunity, and so in the meantime extreme care in the selection of patients is essential.

The Absorption and Fate of Administered Iodine.

Iodine may be administered by mouth either as the free form or as an iodide. The iodine then passes into the blood-stream, after absorption from the stomach or intestine, and this uptake is apparently a very rapid process in the iodine-starved patient. All the administered iodine has been absorbed from the alimentary tract within an hour, and only about 1% is lost in the fæces (Hamilton and Soley, 1940; Keating *et alii*, 1947).

The uptake of iodine by the thyreoid depends partly on the amount of iodine already there. It is found clinically that large doses of Lugol's iodine are much more satisfactory than small doses in producing a pre-operative remission. Certainly only a fraction of this iodine is absorbed, and a high level of serum iodine is needed to saturate the gland. Probably for the same reason the administration of radioactive iodine to a patient recently taking natural iodine results in a very poor pick-up of the active iodine by the gland. Administration of radioactive iodine for tracer tests should therefore be carried out in the iodine-starved patient.

Absorption by the thyreoid again commences at an early stage and it has been detected in the gland within twenty minutes of oral administration (Hamilton and Soley, 1940). A normal patient should retain amounts between 15% and 30% of a tracer dose at the end of forty-eight hours. At the same time as the thyreoid has opportunity of assimilating the absorbed iodine the kidneys are actively excreting iodine. Just as the pick-up by the thyreoid varies according to pathological conditions, so the amount of iodine excreted varies with the absorption by thyreoid or other tissues on the one hand and with the excretory capacity of the kidneys on the other. A normal patient should excrete 50% of the administered dose in twenty-four hours and a further similar proportion of the remaining iodine in the next twenty-four hours.

Quimby gives the following scheme of normal thyreoid function:

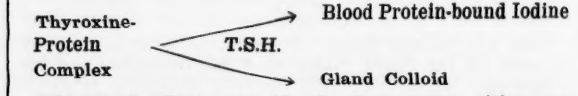
1. Blood. Inorganic iodine passes into the gland under the influence of the thyreoid-stimulating hormone of the anterior lobe of the pituitary, where it is changed to elemental iodine by the action of peroxidase.

$$\text{Blood: Inorganic Iodide} \xrightarrow{\text{T.S.H.}} \text{Gland uptake} \xrightarrow{\text{Peroxidase}} \text{Elemental Iodine}$$

2. Elemental iodine *plus* two tyrosyl groups forms diiodotyrosine; 80% of the fed iodine may be in this form fifteen minutes after the feeding of rats. The thyroxine-protein complex is next formed; 20% of the fed iodine may be in this form within fifteen minutes.

$$\text{Elemental Iodine} \rightarrow \text{Diiodotyrosine} \rightarrow \text{Thyroxine-Protein Complex}$$

3. The thyroxine-protein complex may under the influence of the thyreotropic pituitary hormone pass to the acini to be stored as colloid or may be passed back to the blood as the protein-bound iodine fraction; 90% of the blood iodine is in this form twenty-four hours after the feeding of a test dose.



Thiouracil affects probably the first stages, either preventing the uptake of inorganic iodine by the thyreoid (Rawson's theory) or, by interfering with the peroxidase reaction after its absorption, preventing its return to the elemental state (Chaikoff's theory).

Studies of other tissues after administration of radioactive iodine show that there is no significant accumulation in organs other than the thyreoid. The kidney may be liable to radiation damage from the concentrations produced within it, and similarly the rest of the urinary tract may receive significant dosage. Administration of large quantities of fluids minimizes this risk, which is greatest when therapeutic doses of 30 to 40 millicuries are being administered for carcinoma of the thyreoid. Apparently no immediate renal damage has so far occurred, but this fact does not exclude the possibility that late radiation changes may develop in the future. No effects in the hæmatopoietic tissues are seen, and so anæmia and leucopenia do not occur even after ingestion of large or repeated doses.

TABLE I.

Percentage Uptake in Thyreoids of Children at the End of Forty-eight Hours after Test Dose of I^{131} (Quimby).

Normal Subjects.		Abnormal Subjects.	
Age. (Years.)	Percentage Uptake.	Abnormality.	Percentage Uptake.
<1	12±4	Cretins	<1
1 to 4	10±3	Myxœdema	0
5 to 14	13±3	Hyperthyreoidism, Graves's disease ..	35 to 60
Average ..	12±4	Colloid adenoma ..	3

Pick-Up of Iodine by the Thyreoid.

Quimby and McCune (1947) have investigated iodine pick-up in children and have shown that a useful test is available as an alternative to the difficult basal metabolic test. A tracer dose of six microcuries per stone is administered. Normal children show pick-up, with radioactive iodine of the order of 10% to 15% remaining in the gland at the forty-eight hour stage. Cretins and myxœdematous patients show results in the 0% to 1% range, and hyperthyreoid patients show figures of 30% to 60%.

In adults very similar results were obtained, though the normal thyreoid shows greater retention of iodine and normal patients should show amounts of 15% to 30% remaining in the gland at the end of forty-eight hours.

Keating *et alii* (1947) have studied renal excretion in normal subjects and in patients with thyreoid disease. They conclude that normal people excrete 50% to 60% of administered radioactive iodine within twenty-four hours. They have plotted excreted amounts against time and obtained differently shaped curves for the normal and the various pathological states. After twenty-four hours the normal curve flattens to a plateau. A carcinomatous patient with a slightly smaller thyreoid pick-up has a curve of excretion only slightly lower than the normal; in the myxœdematous patient the excretion rate is higher and the curve does not flatten to a plateau; the excretion rate in hyperthyreoid disease is much less, and so the

excretion curve flattens into a plateau more quickly and at a lower level than for the normal person.

These authors have investigated the problem mathematically and carry out four determinations. They have advanced to the stage at which thyreoid pick-up and renal excretion rates can be stated in terms independent of variations due to altered activity of either organ.

Work with Iodine in Melbourne.

In May, 1948, a small amount of radioactive iodine (I^{131}) was received in Melbourne, and only very short notice of its anticipated arrival was received. The small amount available dictated the policy that tracer tests only could be carried out. The decision was therefore made to undertake such tests with the primary object of developing a reliable technique, which would produce results comparable with those of Quimby and other workers in the various types of thyreoid disease.

A person with a normal neck was first selected and a positive cast was made in paraffin of the shoulders, neck and lower part of the head. The paraffin model was then divided into two parts with a transverse cut passing through the lower part of the neck, and from anatomical charts the positions corresponding with those occupied by the lateral lobes of the thyreoid were found. Holes for two small glass phials were made at these sites. The phials were filled with a known quantity of I^{131} and were sealed. Tests were then carried out with the phials *in situ* in order to calibrate the Geiger-Müller counter placed in suitable positions in front of the model. By this means it was possible to obtain readings which would allow subsequent quantitative estimations of the amount of radioactive iodine in a patient under conditions of a tracer test study.

TABLE II.

Thyreoid Gland Uptake and Urinary Excretion of I^{131} (Quimby).

Glandular State.	Average Percentage in Gland at End of Twenty-four Hours.	Average Percentage Excretion in Twenty-four Hours.
Normal thyreoid	21	52
Toxic goitre:		
(a) Diffuse	64	17
(b) Nodular	52	—
(c) Exophthalmic	57	22
Non-toxic nodular goitre ..	19	50
Hypothyreoidism	3	62
Carcinoma of thyreoid	14	58
Acromegaly (hyperpituitarism) ..	18	53
Symond's syndrome (hypopituitarism)	11	70

The Geiger-Müller counter initially was mounted on a small traversing rack screwed to the bench, and the patient was comfortably seated, with the head fixed in a head clamp, in front of the counter. This set-up had been hurriedly contrived by the Commonwealth X-Ray and Radium Laboratory technical staff so that the tracer tests could be carried out before the iodine became inactive, and proved reasonably convenient for the testing of the thyreoid. But difficulty was encountered in the monitoring of other areas of the patient's anatomy.

The counter has since been mounted on an X-ray stand and satisfactory universal movements are now obtainable. This new suspension was used in the series of tests carried out with the second sample of radioactive iodine to be received in Melbourne.

All the urinary output of the patients was collected in twenty-four hours specimens. On the suggestion of Professor V. M. Trikojus, of the Biochemistry School of the University of Melbourne, sodium hydroxide in solution was added to make the urine alkaline, and then the urine was concentrated by evaporation to reduce its volume to amounts suitable for monitoring. As a test, a known quantity of radioactive iodine was added to a quantity of urine and concentrated by the above means, and subsequent estimation on the Geiger-Müller counter gave a result which agreed very reasonably with the added amount of radioactive iodine. We therefore felt confident that our estimations of renal output were reasonably accurate.

For genetic reasons only elderly patients were chosen for tests. These included patients being treated for other diseases to act as normal controls, patients with thyreotoxicosis of various types, and patients who had received previous treatment, usually operation and post-operative radiation therapy, for carcinoma of the thyreoid. No patients had recently been having iodine medication. Determinations of basal metabolic rate were carried out on all patients for control purposes. Several patients with carcinoma, who had enlarged cervical glands, were tested, but in only one instance were we able to demonstrate any pick-up by any known metastatic deposit. Two patients, who clinically appeared to be thyreotoxic, but whose basal metabolic rate was low, were subjected to tracer tests and high thyreoid iodine-retention figures were obtained in each instance, confirming the clinical diagnosis of toxicity.

The γ-ray emission by I^{131} makes it a dangerous substance to handle, and its mode of transport has received much thought from American authorities. The story of the initial shipments to Australia is a sad one, as two samples wandered about the Pacific on cargo steamers for such a length of time that almost complete decay of activity had occurred before the samples were delivered. Dr. Oddie, of the Council for Scientific and Industrial Research, has designed a special container, which gives adequate protection, occupies reasonably small space and is of such weight that its carriage by air is practicable. But as yet only small amounts of iodine can be made available, and so tracer tests will be continued. It is hoped to obtain sufficient iodine to undertake treatment in a few suitable cases of thyreotoxicosis, and preparations are already in hand for such a therapeutic test. It should be possible to obtain amounts of iodine for a very few such tests, but it seems unlikely that any large scale treatments can be undertaken, firstly on account of protection difficulties in the transporting of the required amount, and secondly on account of the cost, which is quite considerable and must be met in dollars. Unfortunately decay in transit is a dead loss and the amount ultimately received is only about 50% of that originally forwarded.

Radioactive iodine in amounts of the order of four millicuries is used in treatment of thyreotoxicosis, and nearly ten times this amount may be used in the treatment of a suitable case of carcinoma. There appears to be little hope of obtaining sufficient supplies for this last-mentioned purpose.

In association with Dr. Oddie, who has carried the major burden of this tracer work, further tests are planned and treatment of suitable patients will be undertaken as opportunity offers.

The method of evaporating the twenty-four hour samples of urine to a concentrate has proved satisfactory and tests have shown that practically all the iodine in the urine is recovered for measurement. But the evaporation of such a large quantity to small dimensions is not a desirable method, and it is proposed to follow a micro-method used by Keating *et alii* (1947) in future work. The twenty-four hour specimen is made alkaline and a small amount of carrier iodide is added. An amount of 0·2 millilitre is pipetted onto a copper planchette, a small amount of silver nitrate is added, and the specimen is evaporated to dryness. Counts are then made under the window of a Geiger-Müller counter. This method seems simpler, but it must be mentioned that all these monitoring tests take a great deal of time and care.

Oddie is engaged on an extensive mathematical investigation of the rate of absorption and conversion of iodine as it passes through its various possible stages after absorption into the blood-stream, whence it may be excreted or be absorbed into the thyreoid and perhaps undergo alteration into organic form. A comprehensive

series of thyreoid uptake and renal excretion tests has been planned as part of this investigation and work is being continued along these lines as iodine from time to time comes to hand.

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THE TREATMENT OF GRANULAR PROCTITIS WITH "BENADRYL".

By T. EDWARD WILSON, F.R.C.S. (England), F.R.C.S. (Edinburgh), F.R.A.C.S., F.A.C.S., M.R.A.C.P.,

Sydney.

GRANULAR PROCTITIS is also referred to under the names of simple, non-specific, catarrhal, or hæmorrhagic proctitis (or proctocolitis). It usually affects young adults whose general condition is surprisingly good. The chief complaint is of frequent small motions, some of which are well formed, but many consist entirely of blood, pus and mucus. There is a close relationship between the onset and exacerbations of this disease and emotional upsets; and exacerbations are further aggravated by worry over the passage of blood. Milligan[1] has described the appearance of the rectum as "granular, vivid pink and moist. It easily bleeds with trauma, and the normal branched blood vessels are no longer visible, shallow pitting may be present, but deep ulceration of mucous membrane is absent. Discharge on the surface varies from a trace of blood to large quantities of pus, muco-pus and blood". The appearances are beautifully illustrated in his paper. These changes commence in the lower third of the rectum and extend upwards. When improvement occurs they regress in the reverse direction.

While its diagnosis rests on the history and proctoscopic findings, together with the elimination of dysenteric and helminth infections by the pathologist, and presents no problem, granular proctitis remains one of the diseases in which treatment is usually disappointing.

After their introduction new therapeutic agents which have a wide range of activity and which are relatively innocuous are usually soon given a trial in conditions which have previously proved unresponsive to treatment. Thus, as was to be expected, the various sulphonamides have been tried in granular proctitis, each with fresh hope on the part of the patient and of the proctologist, but these hopes have not been sustained.

Granular proctitis has been recognized for decades, but its ætiology is still obscure and there is as yet no specific treatment. Because of this and because in some cases a seasonal variation is exhibited or aggravation is caused by certain items of the diet (Gabriel[2]), "Benadryl" was tried in a series of cases. Many of the patients had already received a variety of treatments including: (i) various sulphonamides given by mouth; (ii) rectal washouts with potassium permanganate, silver nitrate, "Hazeline", and sulphathiazole; (iii) retention enemata of olive oil, bismuth subgallate in olive oil, starch and opium, and sulphathalidine; (iv) suppositories containing mercurochrome, glycerine and ichthyol; (v) local applications to the rectal mucosa of 2% mercurochrome solution; (vi) non-specific treatment, such as low residue diet, extra vitamins, transfusions, tonics, paraffin, kaolin, charcoal, sedatives, "Normocol", "I-so-gel", and rest in bed. Other methods of treatment advised by various authors include: (i) ichthyol and salol given by mouth; (ii) rectal instillations of adrenaline, iodoform, "Argyrol" and ichthyol in olive or cod-liver oil; (ii) irrigations with 2% thymol solution, 1 in 2000 flavine solution, zinc sulphate, copper sulphate, sodium bicarbonate, resorcin, cold water, hot water (45° to 50° C.), 0·5% carbolic acid solution, 5% boracic acid solution, and hydrogen peroxide; (iv) insufflation with "Dermatol"; (v) suppositories containing opium, iodoform, tannic acid, sulphanilamide and sulphasuxidine; (vi) applications directly to the rectal mucosa of up to 10% silver nitrate solution, 25% "Argyrol" solution, and fuming nitric acid; and (vii) protein shock, vaccines, antisera, and ionization with zinc sulphate.

Results.

"Benadryl" is usually dispensed in capsules containing 50 milligrammes of the drug. In this series of 38 cases of granular proctitis one capsule was given three times a day for periods of two to eighteen weeks. An attempt was made to reassure all patients. Those worried by the bleeding were given suppositories containing 2% of mercurochrome to disguise it. If they were constipated, paraffin was prescribed. Other sedatives were not given with the "Benadryl".

An analysis of the patient's subsequent progress shows that in 20 of the 38 cases the symptoms were relieved and in 15 there was considerable improvement in the rectal mucosa. Nearly all patients stated that they felt better after starting the treatment with "Benadryl", but this subjective improvement was not always maintained. In this disease a similar subjective improvement is usually claimed by the patients after the introduction of most new methods of treatment; and in a few cases in such circumstances there may even be, temporarily, a considerable improvement or complete subsidence of the symptoms and of the changes in the rectum.

Case Report.

The following is a summary of the history of a typical patient with a satisfactory response.

A housewife, aged forty-three years, had suffered from granular proctitis on and off for the past nine years. During that time she had been treated with kaolin, paraffin, sulphanilamide, sulphaguanidine, potassium permanganate wash-outs, swabbing with mercurochrome, and a low residue diet. The periodic remissions appeared to be independent of the type of treatment employed.

In September, 1947, she was again complaining of the frequent passage of blood and mucus. On examination the rectal mucosa was red, granular and friable. One capsule of "Benadryl" was given three times a day for twelve weeks, and a steady improvement in the signs and symptoms was observed. The passage of blood and mucus ceased within six weeks of the patient's starting "Benadryl" therapy, and after another ten weeks the rectal mucosa appeared normal.

Discussion.

Exacerbations of granular proctitis usually follow mental stress and worry, and thus sedation has a place in the treatment of this disease. Sedation, though variable

in degree, is one of the main side-effects of "Benadryl", and from this effect alone some improvement in the symptoms would be expected. Also the antispasmodic action of "Benadryl" on smooth muscle should be beneficial in severe cases of this disease. The problem of assessing the value of "Benadryl" as an antihistamine drug in granular proctitis is therefore complicated by the existence of these two side-effects.

In this series of cases the benefits obtained by "Benadryl" therapy were probably greater than those which would have been due to the sedative and antispasmodic effects of the drug or to spontaneous remissions; but it was not possible to prove this. It follows that it was not possible to deduce that granular proctitis is an allergic condition in which "Benadryl" exerts an antihistamine effect. Perhaps larger doses of "Benadryl" would have produced more striking results. However, sedation with other sedatives does not appear to be as effective in the treatment of this disease as "Benadryl", and since it is not impossible that an antihistamine effect played some part in the improvement it would seem that "Benadryl" and other antihistamine drugs are worthy of further trial. A general discussion of the present position of the antihistamine drugs will be found in a recent paper by Burn.[3]

Rest in bed will by itself result in improvement in most patients with granular proctitis, and it is not a great disadvantage if sometimes "Benadryl" enforces such rest. In only one case in this series did the patient (a bus driver) suffer from drowsiness to the extent of impairing his judgement and interfering with his work. This extreme drowsiness could have been obviated by the administration of caffeine and amphetamine, but he was unwilling to continue treatment.

Patients with granular proctitis who suffer from nausea may find it increased when they first take "Benadryl" and usually wish to discontinue the drug even though it is explained that tolerance may develop with subsequent improvement in the patient's condition.

Unlike the sufferers from thyreotoxicosis, who generally are worried over unimportant matters, patients with granular proctitis often have sufficient cause to be very worried about their families or other important problems, and until these problems have been settled little change in the condition of the bowel is to be expected.

Surgical treatment of local complications, such as fissures, hæmorrhoids and hæmatomata, should be avoided whenever possible, for, as Gabriel[2] points out, "In the presence of a proctitis the healing of anal wounds is likely to be slow and perhaps unsatisfactory".

Conclusions.

"Benadryl" is sometimes of value in granular proctitis and, in the present state of our knowledge, its use is advised.

In addition to "Benadryl" a diet of low carbohydrate, low roughage and high vitamin content, avoidance of overwork, warmth, and antisymptomatic remedies are indicated. The antisymptomatic remedies include paraffin if the patient is constipated, iron if he is anæmic, mercurochrome suppositories to disguise persistent bleeding which is causing anxiety and to pander to the patient's desire for local treatment, and kaolin.

Acknowledgement.

These patients were treated at Saint Mark's Hospital, London, while I was a resident surgical officer of that hospital; I am greatly indebted to the honorary staff and especially to Dr. W. B. Gabriel for continued help and advice.

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RUBELLA CONGENITAL INNER EAR DEAFNESS IN TASMANIA.[1]

By B. HILLER,

Honorary Ear, Nose and Throat Surgeon, Royal Hobart Hospital, and Honorary Aurist to the Tasmanian Institute for the Blind and Deaf, Hobart.

IN 1941 Gregg[1] published his article establishing the fact that cases of congenital cataract were resulting from rubella occurring in the mother during the first four months of pregnancy, and subsequently a greatly increased incidence of congenital inner ear deafness as well as of cardiac and other defects was soon ascribed to the same cause. Much work has since been done on the whole subject by Swan, Tostevin and their associates,[2][3][4] and by Carruthers[5] and others. The results of their investigations have been corroborated in other countries where similar but less severe epidemics have also occurred (E. Clayton-Jones[6] in England and C. A. Erickson[7] in America).

The children with congenital inner ear deafness due to maternal rubella occurring in the epidemics between the years 1937 and 1940 inclusive have now reached the age at which they are able to offer full cooperation for examination of their hearing. It is now possible to obtain reliable audiograms and hence more precise information as to the type of hearing defect.

In a personal communication the Commonwealth Director-General of Health, Dr. A. J. Metcalfe,[8] informs me that the Australian Capital Territory was the only territory in the Commonwealth in which rubella was a notifiable disease at the time of the epidemics mentioned above. Table I shows the statistics for the relevant years,

TABLE I.

Year.	Rubella Deafness Cases in Australian Capital Territory.	Rubella Deafness Cases in Tasmania.
1935	Nil	Nil
1936	Nil	Nil
1937	158	Nil
1938	6	12
1939	1	1
1940	504	2
1941	23	17

1935 to 1941 inclusive, together with the incidence of rubella deafness in the Tasmanian Institute for the Blind and Deaf. It will be seen that the rubella deafness peak years of 1938 and 1941 correspond very closely to the peaks of the rubella epidemic in the Australian Capital Territory—namely, 1937 and 1940.

But the peaks of maternal infection, according to the investigations carried out in South Australia by Swan, Tostevin and Black,[4] were in 1939, 1940 and especially 1942. In New South Wales the peak year was decidedly 1940. (See the report of a committee appointed by the Director-General of Public Health of New South Wales.[9]) It is therefore apparent that the peaks of incidence of rubella infection in various parts of Australia were not concurrent.

Review of Congenital Inner Ear Deafness at the Tasmanian Institute for the Blind and Deaf.

In 1948, when this review was made, there were at the Tasmanian Institute for the Blind and Deaf 32 children with rubella congenital deafness as well as 10 with non-rubella congenital inner ear deafness. These numbers include the vast majority of all such children born inclu-

[1] Read at the annual meeting of the Tasmanian Section of the Ophthalmological Society of Australia (British Medical Association) on March 19, 1949.

sively between 1932 and 1945 in Tasmania. Two children who have a complicating middle ear deafness have been excluded from the non-rubella deafness group. There has been a notable absence of middle ear complications in the rubella group.

As seen in Table II, the 32 rubella deaf children were born in the years 1938 to 1941 inclusive—12 in 1938, one in 1939, two in 1940 and 17 in 1941. Results of examination of the *fundi oculorum* detailed in an article by J. B. Hamilton, F. Phillips, C. R. Palfreyman and D. H. Waterworth[10] have shown that five of the 1938 group of 12 have rubella retinitis, whilst there are ten amongst the 17 children born in 1941. It should be stated here that of the five deaf children with retinitis born in 1938, four of the mothers denied the occurrence of rubella in early pregnancy, and of the 10 deaf children with retinitis born in 1941 two mothers gave no history of rubella during the pregnancy. It follows that these six cases in all would have been classified as non-rubella congenital deafness, if it had not been for the presence of characteristic rubella retinitis.

TABLE II.
Classification of Rubella Congenitally Deaf Children.

Group.	Year of Birth.				Totals.
	1938	1939	1940	1941	
Rubella - deaf children:					
Male ..	3	1	1	9	14
Female ..	9	—	1	8	18
Total ..	12	1	2	17	32
Children with retinitis:					
Male ..	2	—	—	6	8
Female ..	3	—	—	4	7
Total ..	5	—	—	10	15
Children with congenital cardiac defects:					
Male ..	—	1	—	—	1
Female ..	—	—	—	—	—
Total ..	—	1	—	—	1

There have been no admissions to the institution of rubella-deaf children born later than 1941.

I am indebted to Dr. J. P. Millar[11] for the results of his examination of the hearts of all patients, and he informs me that there is only one, amongst all the rubella-deaf children, who has a congenital cardiac defect—a boy, born in 1939, who has aortic stenosis. There are no other congenital defects of any description.

Types of Audiograms.

All the children in the rubella and non-rubella congenital deafness groups, 42 in all, have had their air-conduction hearing losses carefully tested by means of a correctly calibrated pure-tone audiometer lent by the Commonwealth Acoustic Research Laboratory. The readings were taken for each of five frequencies at intervals of an octave commencing at 256 cycles per second (middle C on the piano), the highest frequency being 4096 cycles per second.

In audiometry, the hearing loss is measured in units called decibels. A decibel is a unit of relative intensity (W. L. and H. C. Ballenger[12]), and is the smallest increase in sound appreciable by the normal ear. It is a logarithmic unit in which 10 decibels would correspond to a tenfold increase in sound intensity, 20 to a hundredfold, 30 to a thousandfold and so on.

Figure I shows an audiogram of a subject with a typical inner ear deafness curve. Seven of the non-rubella cases conform exactly to this type of curve whilst in the remaining three the curves are somewhat "flatter". These will be discussed later.

In contrast, the audiograms of the rubella-deaf subjects do not adhere regularly to any one type of curve, and four completely different patterns are seen in Figures II and III, which are audiograms of two patients (H.D. and M.E.) in Table III. Thus, even curves for the right and left ear of the same child differ considerably from each other in each of these two subjects.

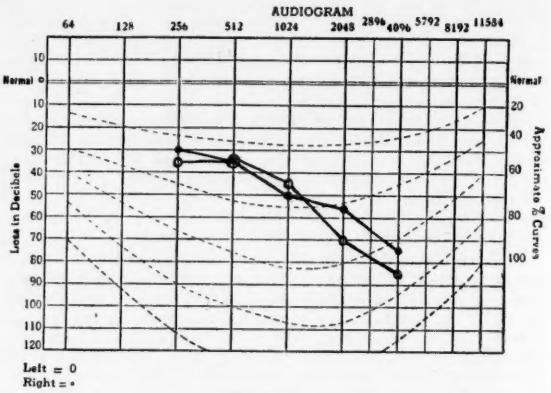

FIGURE I.
Typical inner-ear deafness curve.

The asymmetry between the two ears in a single case may be carried to extremes, as was exemplified in a child examined by me in consultation some twelve months ago.

This child was born in another State in 1940 and had unilateral rubella inner ear deafness. The right ear was totally deaf in the speech range, whilst the hearing in the left ear was perfectly normal.

Similarly, this feature of rubella is also exemplified in the occasional occurrence of monocular rubella cataract, as reported by Gregg[13] and by Day[14] in Australia and by Erickson[7] in America.

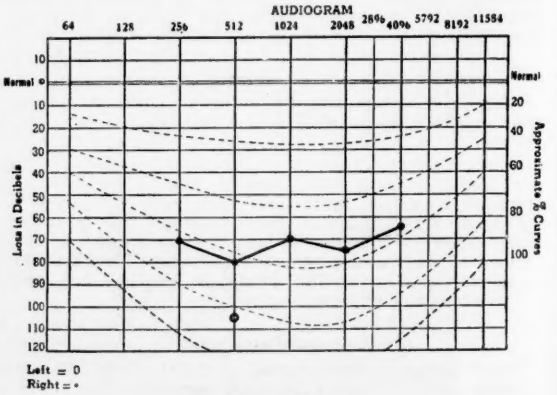

FIGURE II.
A rubella-deaf patient's audiogram (H.D.).

Average Hearing Losses.

The average hearing loss for each subject has been calculated from the figures for the hearing losses over the three octaves between 512 and 4096 cycles per second. Hearing over the speech range lies within these three octaves. The method of striking the average was that used by the Commonwealth Acoustic Research Laboratory—namely, first the average loss within each of the three octaves is

taken and from these the average loss of the three octaves is computed (Murray[35]).

The average loss in decibels in each ear of each of the 32 rubella and 10 non-rubella subjects is shown in Tables III and IV respectively.

Degrees of Deafness.

For the purposes of this review the degrees of deafness have been classified into four grades, and at the Tasmanian Institute for the Blind and Deaf we have found that in actual practice the cases fall into these grades in the proportions shown in Table V.

TABLE III.
Rubella Congenital Deafness.

Year of Birth.	Patient's Name.	Average Hearing Loss (Decibels).	
		Right Ear.	Left Ear.
1938	A.W.	62	64
	T.W.[1]	85	56
	F.W.[1]	98	85
	P.S.	66	85
	B.O.[1]	79	79
	H.G.	75	83
	M.E.	81	97
	L.W.[1]	84	104
	R.S.	95	87
	J.K.	98	96
	A.C.	103	98
	C.D.[1]	105	104
1939 and 1940	P.L.[2]	93	105
	K.T.	102	102
	J.H.	89	87
1941	P.H.[1]	99	57
	D.T.[1]	104	69
	R.A.[1]	52	51
	H.D.[1]	72	(Complete)
	D.D.[1]	67	(Complete)
	R.B.[1]	73	87
	M.E.	67	91
	E.P.	109	91
	N.W.[1]	(Complete)	109
	B.L.[1]	87	88
	D.D.	102	81
	T.B.	109	103
	J.C.L.[1]	(Complete)	(Complete)
	S.C.	(Complete)	(Complete)
	J.M.	105	87
	R.R.	110	(Complete)
	T.R.[1]	105	110

[1] Rubella retinitis present.
[2] Cardiac defect (aortic stenosis) present.

TABLE IV.
Non-Rubella Congenital Deafness.

Name.	Year of Birth.	Average Hearing Loss. (Decibels.)	
		Right Ear.	Left Ear.
B.B.	1932	84	(Complete)
D.T.	1933	(Complete)	(Complete)
B.M.	1936	(Complete)	106
B.S.	1937	100	103
I.C.	1937	89	96
A.L.	1938	94	100
M.C.	1938	100	103
I.B.	1939	79	79
P.W.	1941	78	66
D.W.	1941	82	90

From this it will be seen that 12 out of the 32 rubella subjects are wearing individual hearing aids, and two out of 10 non-rubella children; eight of the rubella-deaf subjects, two of the non-rubella-deaf subjects are suitable only for the group hearing aid. It is found that as a general rule those children who have an average hearing loss of 80 decibels or under in the better ear for tones (or frequencies) in the speech range are capable of deriving benefit from an individual hearing aid, whilst those with an average loss of between 80 and 90 decibels are suitable for the group hearing aid only. Awaiting solution is the important problem of providing sufficiently powerful individual hearing aids for the somewhat large proportion of subjects whose average hearing loss lies between 80 and 90 decibels.

Those with hearing in the speech range but insufficient for modern hearing aids number 10 of the rubella and five of the non-rubella subjects. Those with no measurable hearing in the speech range number two and one respectively in the two groups.

From Table V it is evident, too, that on the whole for practical purposes the deafness in the rubella group is of lesser degree than that in the non-rubella group. If Grades

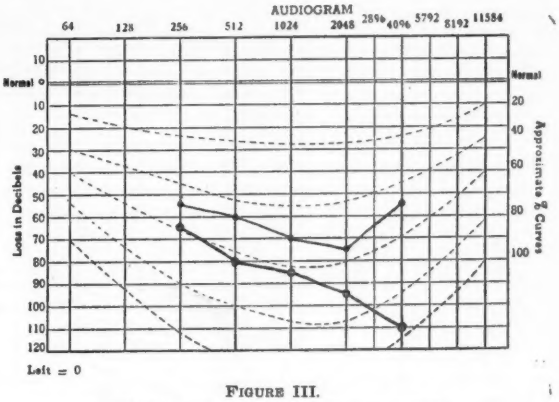

FIGURE III.
A rubella-deaf patient's audiogram (M.E.).

I and II are taken together (those subjects deriving benefit from either an individual or a group hearing aid), 20 of the rubella subjects (out of 32) are, or will be, able to hear and understand the spoken word, whereas of the non-rubella subjects the number is only four out of 10. The education of those belonging to Grades I and II therefore does not present such a difficult problem as does that of those in Grades III and IV.

The first group, which consists of those suitable for individual hearing aids (Grade I in Table V) are now being educated in a special acoustic class attached to a normal State school and taking part in all normal school activities. So far this scheme promises great success. This group commenced their education with the use of a group hearing-aid, which is capable of taking 12 children, each child being provided with earphones and an individual microphone attached to the desk which enables him to hear his own voice. In addition every desk is fitted with a tone and volume control for each child. This obviously is of great assistance in speech therapy.

Although this group aid is still part of the equipment in the special acoustic classroom, the children prefer to use their individual aids continuously. These allow for much greater flexibility in classroom activity.

TABLE V.
Four Practical Grades of Deafness in Congenitally Deaf Subjects.

Grade.	Rubella.	Non-Rubella.
I: Suitable for individual hearing aid—average loss in speech range 80 decibels or under in better ear	12	2
II: Suitable for group hearing aid only—average loss in speech range 80 to 90 decibels in better ear	8	2
III: Hearing in speech range, but insufficient for modern aids	10	5
IV: No measurable hearing in speech range—totally deaf	2	1

The individual aids are worn with the control turned to "on" throughout the day, and the children wear them without self-consciousness.

Using another group aid in the deaf school proper is a group of children with a loss of between 80 and 90 decibels approximately—that is, Grade II—and included with these are some of Grade I, who, although they have hearing within the speech range, have proved as yet mentally incapable of learning in a special class with an individual aid.

A third group of children, which includes Grades III and IV of the classification, with a hearing loss too great

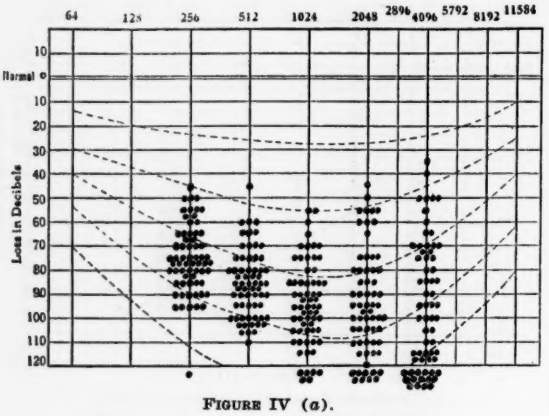

FIGURE IV (*a*).
Type I: All rubella-deaf subjects (32).

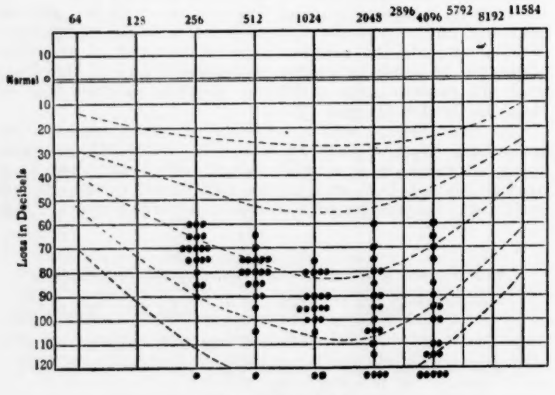

FIGURE IV (*b*).
Type II: Non-rubella congenitally deaf subjects (10).

for any type of aid to reach them, are taught by the combined use of oral work, lip-reading and the manual alphabet.

The average intelligence of the rubella-deaf children, according to the teaching staff, would appear to be the same as that of a cross-section of any similar group of hearing children; that is, a few are very intelligent, most are of average intelligence and a few are subnormal.

There are many obvious disadvantages in keeping the children, especially those with useful hearing, in the environment of a deaf community, as the child must as far as possible learn to take his place among hearing people. Therefore the policy always is to give the child as much of his own home life as possible.

Unfortunately, owing to the wide distribution of their homes throughout the State, many children must of necessity be full boarders during the school terms.

Comparisons Between Various Types of Cases.

Figures IV, V and VI are composite audiograms showing hearing losses in six types of cases. The types are coupled together in three pairs for purposes of comparison. Each of these audiograms shows the hearing loss for the various frequencies of sound at intervals of an octave for every ear in the type indicated. The dots below the graphs indicate that hearing for the particular frequency is either non-existent or beyond the limits of the audiometer.

It will be seen throughout that the general tendency is for the hearing loss in the rubella cases to be least for the low tones and to increase slowly and steadily octave

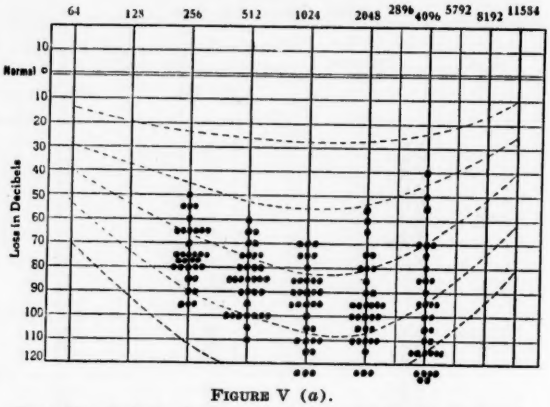

FIGURE V (*a*).
Type III: Rubella deafness without retinitis (17 subjects).

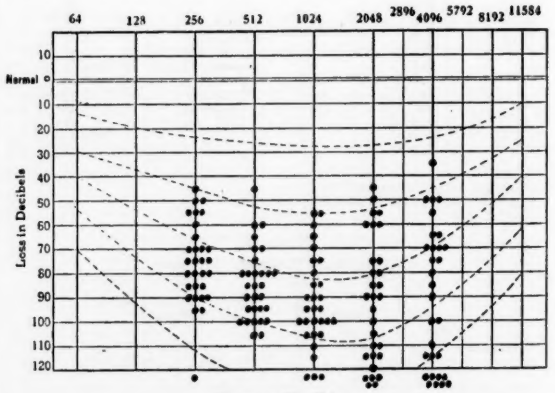

FIGURE V (*b*).
Type IV: Rubella deafness with retinitis (15 subjects).

by octave as the sound frequency or pitch rises until 2048 cycles are reached. At this point in a number of cases there is a tendency for the loss to decrease as the pitch rises, but in many of them the tendency to increasing loss continues. As a result all the composite audiograms of rubella deafness subjects show in the two higher pitches an increasing tendency of the readings to spread out up and down the vertical lines indicating the number of cycles per second.

Table VI is a table of comparisons between the over-all averages of the average individual hearing losses in the various types mentioned. Examination of the data in this table as well as of the composite audiograms (Figures IV, V and VI) gives the following information concerning the six types.

1. Types I and II: Rubella-deaf subjects have less hearing loss than non-rubella-deaf subjects. The former (32 subjects) show less hearing loss than the latter (10 cases)—85 decibels average loss in the good ear, 99 in the

worse ear and 92 in both ears, as against 91, 100 and 95 decibels respectively. These figures, together with the grading in Table V, already discussed, agree with Murray's findings (15) that the average degree of deafness in the rubella-deaf subjects is less than that in the non-rubella-deaf subjects, although the difference is not great. Hence comparison of the two composite audiograms in Figure IV shows no obvious difference in the general picture.

2. Types III and IV: Rubella subjects with retinitis show less hearing loss than those without retinitis. Fifteen subjects with retinitis have an average loss of 81 decibels in the better ear, 99 in the worse ear and 90 in both ears,

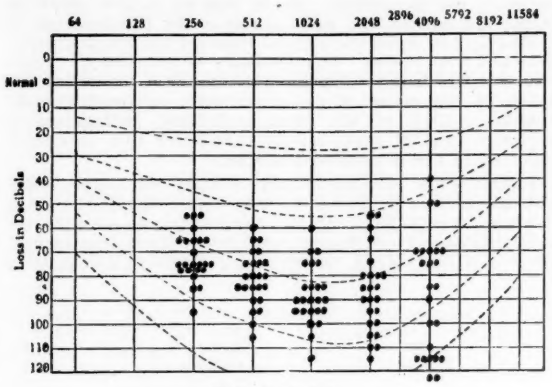

FIGURE VI (*a*).
Type V: Rubella-deaf subjects born in 1938 (12).

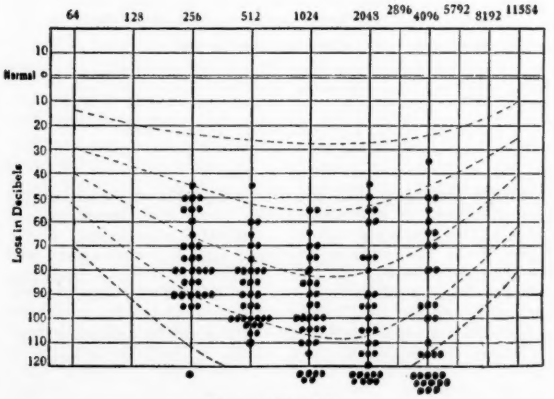

FIGURE VI (*b*).
Type VI: Rubella-deaf subjects born in 1941 (17).

whereas 17 subjects without retinitis have an average loss of 89, 99 and 94 decibels respectively. The composite audiogram in Figure V shows a slight general "shift" upwards in the retinitis cases (indicating less hearing loss), as compared with the non-retinitis cases. Thus, the rubella subjects with retinitis have slightly less hearing loss than those without retinitis.

3. Types V and VI: Rubella-deaf born in 1941 have greater hearing loss than those born in 1938. Seventeen patients born in 1941 have an average loss of 86 decibels in the better ear, 104 decibels in the worse ear and 95 decibels in both ears, whereas the 1938 cases have corresponding figures of 81, 91 and 86 decibels respectively. Again, the composite audiogram, Figure VI, demonstrates a general "shift" downwards for the 1941 cases, as compared with those of 1938. Thus it is evident that Tasmania's rubella-deaf born in 1941 have greater hearing loss than those born in 1938.

Relative Severity of the 1938 and 1941 Rubella Cases in Tasmania.

After careful investigation of the data, the following facts emerge, indicating that the maternal rubella outbreak causing the 1941 epidemic was decidedly more devastating than that responsible for the epidemic of 1938.

1. More deaf children were born in 1941 than in 1938—17, compared with 12 in 1938.

2. The deafness in the 1941 group was of greater severity, as has just been shown.

3. Of all rubella-deaf subjects in all years, there are two only who have bilateral total deafness in the speech range, and they are both in the 1941 group. Four other subjects each have one totally deaf ear—all in the 1941 group. That is, a total of eight ears in the 1941 group of 34 ears is totally deaf. There are no ears whatever in any of the other relevant years which have no measurable hearing in the speech range.

4. The proportion of retinitis is greater in the deaf subjects born in 1941 (10 out of 17, as against five out of 12 born in 1938).

Possibly, too, the severity of symptoms in the epidemic of rubella causing the 1941 cases of deafness was greater than that in the epidemic responsible for those of 1938. This is suggested by the fact that in 1938 no less than four out of 12 deaf subjects had no history of maternal rubella, and their deafness was diagnosed as due to rubella only by the presence of retinitis, whereas the proportion for 1941 was only two out of 17 subjects.

TABLE VI.
Average Losses in Hearing.

Type of Case.	Number of Subjects.	Average Loss in Better Ear. (Decibels.)	Average Loss in Worse Ear. (Decibels.)	Average Loss in Both Ears. (Decibels.)
1. All rubella deafness ..	32	85	99	92
2. Non-rubella congenital deafness	10	91	100	95
3. Rubella deafness without retinitis	17	89	99	94
4. Rubella deafness with retinitis	15	81	99	90
5. Rubella deafness (subjects born in 1938) ..	12	81	91	86
6. Rubella deafness (subjects born in 1941) ..	17	86	104	95

Average Hearing Loss of Rubella-Deaf Subjects in Tasmania.

Reference to Table VI shows that in Tasmania the rubella-deaf subjects have an average loss over the speech range of 85 decibels in the better ear, 99 decibels in the worse ear and 92 decibels in both ears. Murray[GB] in New South Wales, working on 105 cases from all States of the Commonwealth, discovered much less average hearing loss, his figures being 65 decibels in the better ears and 72 decibels in both ears.

Analysis of the Non-rubella Congenital Inner Ear Deafness Cases.

I believe that it may be interesting now to survey the non-rubella inner ear deafness subjects in the Tasmanian Institute for the Blind and Deaf. There are ten of these (see Table IV), one born in 1932 (unilateral total deafness), one in 1933 (bilateral total deafness), one in 1936 (unilateral total deafness), two in 1937, two in 1938, one in 1939 and two in 1941. Thus in this group there are five subjects born in the same period as all the rubella-deaf subjects under consideration in this review (two born in 1938, one in 1939 and two in 1941).

We have already seen that among the 12 rubella-deaf subjects in 1938 the deafness of four was diagnosed as due to maternal rubella solely by the presence of pathognomonic rubella retinitis. As 17 subjects out of a total of 32 for all years of all the rubella-deaf do not show retinal changes, it is reasonable to conclude that the probability

is that some of the group of non-rubella congenitally deaf children are in reality rubella subjects—especially as the audiogram of rubella deafness is not characteristic.

P.W. (Case I in Table IV) is one of these. She is classified as a non-rubella-deaf child born in 1941, the year of the greatest incidence of rubella deafness. She has a congenital cardiac condition diagnosed as interauricular septal defect. In all probability she is also a rubella subject, especially as her father positively suffered from rubella in the first month of his wife's pregnancy. How mild his wife's infection must have been is shown by the fact that she is emphatic that she never had a rash during pregnancy and says she has never had rubella at any other time. Nevertheless, there is the great probability that the mother of the child had a mild and unrecognized rubella infection in the early months of her pregnancy, and further, that its effects were such as to cause in the infant congenital deafness (not extreme—66 decibels average loss in one ear and 78 decibels in the other) as well as an interauricular septal defect.

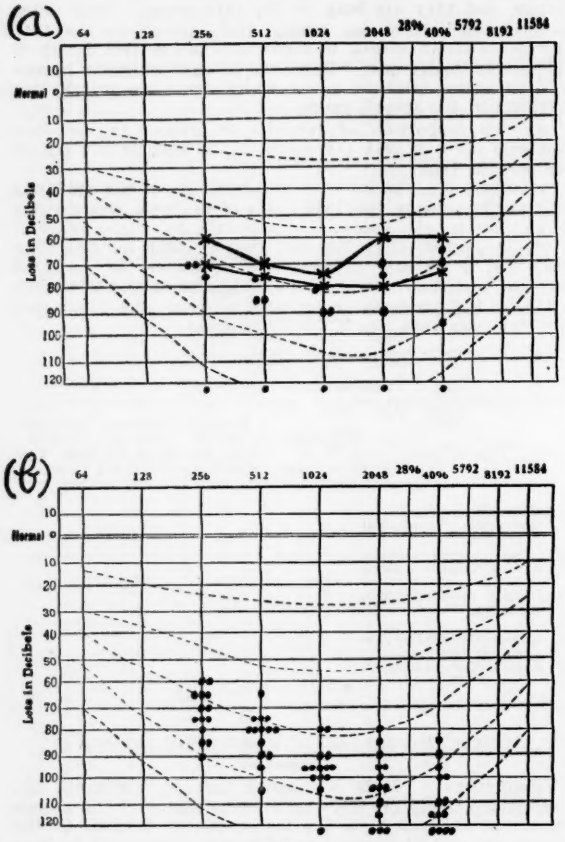

FIGURE VII.
Subdivision of the ten so-called non-rubella-deaf subjects: (*a*) possibly rubella-deaf (three); (*b*) non-rubella-deaf, probably hereditary (seven).

It is fair to ask whether there are not other true rubella deafness cases which are at present classified amongst those of non-rubella congenital inner ear deafness.

On examination of the individual audiograms of the group of 10 non-rubella-deaf subjects, seven of them are found to have remarkably consistent and typical inner ear deafness audiograms; this seems unlikely to be merely coincidence. But the other three are completely atypical and have somewhat "flat curves", that is, more or less horizontal. One of these is P.W., whose case has just been discussed, and who is almost certainly a rubella subject and was born in a "rubella year" (1941). I.B. (Case II) was born in 1939, one of the two years intermediate between the two peak "rubella years", a year in which only one proven rubella-deaf subject was born. B.B. (Case III) was born in 1932, not a "rubella year"; but he has congenital absence of the auditory canal on the left side with malformation of the auricle on both sides.

Possibly, then, all three of these children with audiometric curves not typical of the usual inner-ear deafness curve may perhaps be subjects of maternal rubella infection.

Figure VII (*a*) shows a composite audiogram of these three possible rubella subjects. The crosses connected by lines are the readings of P.W. (Case I, which is almost certainly one of rubella). Again, you see in this graph, as mentioned earlier, the same tendency for the readings to spread up and down the vertical lines for the frequencies of 2048 and 4096 cycles. This tendency is not seen in Figure VII (*b*), which is a composite audiogram of the remaining seven of the group of ten non-rubella congenitally deaf subjects. Each of the seven individual audiograms is typically that of inner-ear deafness, and this is clearly reflected in the composite audiogram.

Examination of the family history of these seven patients reveals the fact that three have a family history of deafness (see Table VII), and the parents of a fourth are first cousins. Thus, out of seven cases there remain only three in which there is no ascertainable probable hereditary factor.

TABLE VII.

Patient.	Family History of Congenital Deafness.
D.T.	Sister deaf: grandparents first cousins.
B.M.	Nil.
B.S.	Two brothers and two uncles deaf.
I.C.	Uncle and brother deaf.
A.L.	Nil.
M.C.	Nil.
D.W.	Nil, but parents are first cousins.

Whilst realizing that the small number of cases in the two divisions of the non-rubella cases are insufficient to permit the drawing of any definite conclusion, I put forward the following suggestion: that the audiogram of a congenital inner-ear deafness subject without a maternal rubella history showing a curve not conforming to the typical inner-ear deafness curve may perhaps be diagnostic of deafness caused by damage to the fœtus resulting from rubella. Or, perhaps more likely, it may eventually be shown that congenital deafness, hereditary in origin, has a typical inner-ear deafness curve, and that all atypical curves in congenitally deaf subjects may indicate any other causal factor at present known or unknown.

Stage of Pregnancy.

Before I conclude this paper, let me say a few words on my attempt to ascertain the exact stages of pregnancy at which the mothers incurred the rubella infection. All were somewhat vague and not one was able to give the exact week. The times given by the 26 mothers are seen in Table VIII.

The indefinite nature of the dates can readily be understood, because usually it is not until the child is about three years of age that the parents become fully aware that the child is really deaf. When the aurist is consulted, the mother is then asked to date an event which occurred at least some three and a half years previously. Naturally, she finds this difficult and her answer is somewhat indefinite.

In attempts to ascertain the exact stage of development of the fœtus at the time of infection of the mother, a further uncertain factor in the vast majority of cases is

the inability to determine the exact date of conception. It is, therefore, frequently somewhat of a conjecture when we state that it was at a certain week in the period of pregnancy that the inner ear was attacked by the virus of rubella.

TABLE VIII.

Maternal Rubella—Stage of Pregnancy.

Group.	First Month.	Second Month.	Third Month.	Early Pregnancy.	Fifth Month.	Stage Not Remembered
Retinitis ..	1	2	4	2	—	—
Without retinitis	1	3	8	3	1	1
Total	2	5	12	5	1	1

Summary.

1. Forty-two congenitally deaf children in the Tasmanian Institute for the Blind and Deaf have been examined.

2. The deafness in 32 cases was due to rubella occurring in the mother in the early months of pregnancy, and the other 10 are classed as non-rubella inner-ear deafness cases.

3. An analysis of the degrees of deafness and of the types of audiometric curves of various groups of children has been made.

4. The average degre of deafness in the rubella-deaf group is less than that in the non-rubella cases.

5. Rubella patients with retinitis show less average hearing loss than those without retinitis.

6. Rubella-deaf children born in 1941 have greater average hearing loss than those born in 1938.

7. The non-rubella congenital inner-ear deafness cases have been analysed, and reasons are advanced for believing that a proportion of cases so classified at present may have resulted from rubella infection in the mother in early pregnancy.

8. The suggestion is put forward that the clue to the causal factor in congenital inner-ear deafness may lie in the audiogram, and that the deafness of patients with typical inner-ear deafness curves is probably of hereditary origin.

Acknowledgements.

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THE RESULTS OF OPERATIVE TREATMENT OF INTERVERTEBRAL DISK PROTRUSION: A FOLLOW-UP REPORT OF 108 CASES.[1]

By DOUGLAS MILLER AND G. K. VANDERFIELD,

Sydney.

PATIENTS are often afflicted by sciatica of intervertebral disk origin for too long before being submitted to surgical treatment. Perhaps the reason is that the clinician is slow to recognize the condition, possibly because of some fears that surgical treatment does not yield good fruits, or may result in a long period of disability. In order to gain information on the latter point we have made a follow-up in a personal series, and have selected from a total of over 300 patients submitted to operation 125 consecutive civilian patients, all engaged in active and often heavy manual work.

In 300 cases there were two deaths, one due to pulmonary embolism and one to the effects of paralytic ileus. In this analysis of results an attempt has been made not merely to emphasize the very satisfactory results of surgery, but to discover a prognostic basis of success or failure in this form of treatment.

It will be seen that the duration of symptoms indicates that intervertebral disk sciatica is not readily a self-righting disorder and that the period of pre-operative invalidism contrasts strongly with the time taken to obtain full post-operative recovery.

The Follow-Up.

One hundred and twenty-five patients were sent *questionnaires*, and replies were received from 108. The times since operation varied from ten years to one year.

The questions asked were as follows: (i) Are you quite free from pain? (ii) If not, where is it? (iii) How long after operation did you return to work?

The replies have been classified into the following groups:

1. Completely satisfactory. The result is classed as completely satisfactory when the reply to the first question is "yes" and the patient has returned to his normal work. There were 54 in this group.

2. Group having minor residual symptoms. Patients are put into this group when the reply to the first question is "no", and the reply to the second question indicates only minor residual or occasional symptoms and the patient has returned to his former work. There were 41 in this group. The residual symptoms described were occasional pain in the leg, back stiffness after exertion and cramps in the legs. These did not interfere with their normal

[1] Read at a meeting of the Royal Australasian College of Surgeons, Brisbane, June, 1949.

activities, and the group includes miners, farmers, a bricklayer, a bus driver and three nurses.

3. Unsatisfactory. The result is classed as unsatisfactory when the reply to the first question is "no", and the second reply indicates a continuance of such symptoms that the patient has been unable to return to his former work because of disability, or because of advice not to do so. There were 13 patients in this group. Ten of these had changed their occupation and three have been unable to work.

An attempt has been made to see if it might be possible in the pre-operative investigation to recognize clinical features which could be characteristic of each group, as well as operation findings indicative of a good prognosis.

All patients who were subjected to operation had in common a severe degree of suffering, resistance to conservative measures and an incapacity to follow their normal occupations.

The actual criteria for operation have been discussed in a previous paper (Miller and Vanderfield, 1947). The operations were of a conservative character, frequently being performed without removal of bone—total disk destruction was never attempted. One fusion only was performed.

The Completely Satisfactory Group.

An analysis of the totally satisfactory group shows that in all cases the symptoms were very severe and there was an unequivocal character in their signs. The average age of patients in this group was thirty-six years, but ranged from nineteen to sixty-two years. The average time lost from work varied from five weeks to ten years. The patient who lost ten years was a farmer who had averaged three months a year in bed for forty years, making a total of ten years' complete invalidism. Two patients had suffered between two and seven years and of the remainder the average period of invalidism had been six months.

The findings at operation were unequivocal. In 44 cases there was a completely sequestrated fragment of disk lying beneath a tightly stretched nerve root; in five the disk was soft and degenerate but not sequestrated. There was one negative finding. In four cases the findings were not recorded. It was common in this group to find a hypertrophied *ligamentum flavum* in association with the gross disk change.

These patients all had a short post-operative convalescence and the time before return to full work varied from five weeks to three months, with an average of eight weeks. Their occupations are in many cases very arduous: one man fells a load of timber every day, others are miners, farmers and truck drivers; one has returned to championship tennis and another to rough-riding.

The Group with Minor Residual Symptoms.

In the second group (consisting of patients relieved of all severe pain but having some minor residual or recurrent symptoms) the age period was the same as in the first group, but the characteristic clinical features had been less severe than in the previous group. On the whole the histories were longer. The findings at operation were not always so striking. Eighteen patients had completely sequestrated disk fragments beneath the nerves, three had no apparent abnormality and 20 had a bulging disk protrusion requiring incision to liberate degenerate contents.

The time of return to work in this group varied from a few weeks to eleven months with an average of between two and three months. The patient taking eleven months had suffered foot drop prior to operation.

The Unsatisfactory Group.

The third or unsatisfactory group is the most important to analyse, for in this group alone could it be argued that the patients were unsuitable for surgery. The age group is higher; the youngest patient was aged twenty-nine years and the oldest sixty years, the average being forty-five years. It is noteworthy that nine had sustained a severe back injury immediately preceding the onset of their disability. Two could be regarded as psychologically "poor risks". Nine were miners. All were seriously disabled, and the physical signs provided convincing evidence of nerve-root damage. The operative findings varied; in no case was there a sequestrated disk fragment. Of the ten patients who are working, five had soft degenerate disks, four had very collapsed spaces with general absorption of the disk and one had no apparent abnormality.

Of the three who cannot work, one had a huge central disk extrusion, and the others a degenerate disk with a collapsed intervertebral space. It should be mentioned that several of these patients included notes with their card expressing gratitude for a considerable degree of relief from their former state. It cannot therefore be said that the labour spent on them is totally lost.

It would not appear an easy matter to recognize preoperatively this type of case, except that the patient is older and there is usually a history of injury. Such experience conforms to experience elsewhere in the traumatic surgery of the locomotor system. It is also to be noted that 10 out of 13 patients were miners drawing compensation. In giving this fact its full value one must remember the strenuous character of mining and the fact that the other groups also include many patients drawing full compensation.

Conclusion.

The general review of this series indicates that the results of this branch of surgery are highly satisfactory and stand favourable comparison with those of any other branch of surgery.

The most striking success comes to the worst type of case. Where a patient is afflicted terribly and where operation discloses a gross pathological state, success is assured. Where the clinical picture is less pitiable and the operative findings less dramatic, complete success is likely to be marred by minor residual symptoms.

Success is not so sure in a small group, most of the members of which are older and are miners receiving compensation. Several patients of this type in our series had suffered a back injury and in none of these were typical disk extrusions disclosed at operation.

Previous follow-up investigations carried out by various surgeons have yielded somewhat similar results.

In 1945, 513 patients operated on by Dandy were followed and good results corresponding to our first two groups were found in 63% of cases; improvement occurred in 20% of cases and no improvement in 17%.

Davis (1947) was able to follow 84; 74% of these were able to return to their former occupation, 21% had changed their occupation.

Geoffrey Jefferson (1948) has discussed 130 patients; 82% of these were cured or had insignificant symptoms.

Summary.

1. Follow-up cards were sent to 125 civilian patients who had been operated upon for intervertebral disk sciatica.

2. One hundred and eight replies were received.

3. Ninety-seven of the patients who replied (90%) had returned to their former occupation; 54 of these were completely free from symptoms and 43 had insignificant residual or recurrent symptoms.

4. Ten patients were unable to return to their former work. Three were unable to do any work.

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Reports of Cases.

THE USE OF STREPTOMYCIN IN THE TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS: REPORT OF A CASE.

By GEORGE V. HALL,
Sydney.

THE successful use of streptomycin in the treatment of subacute bacterial endocarditis has been reported in the literature, but on relatively few occasions.

Owing to the fact that penicillin is the chemotherapeutic agent of choice the use of streptomycin is confined mainly to those cases in which the organism has proved penicillin-resistant. In most of these cases, as indeed in this one, the causative organism has proved to be a pleomorphic streptococcus, generally a streptococcus of unusual type.

Thus Priest and McGee (1946) used streptomycin with apparent success in two cases in which the infecting organism was a pleomorphic streptococcus, the earliest and most pleomorphic forms being Gram-negative and the older forms which appeared as pairs and short chains of cocci being Gram-positive. Later, however, after the culture had been frozen, the organism grew as a typical *Streptococcus viridans* (type not specified) with a given hæmolytic zone. In both cases the organism appeared to be highly resistant to penicillin but sensitive to streptomycin.

Cady and Allen (1946) and Cady and Hunter (1947) reported the unsuccessful treatment of subacute bacterial endocarditis due to *Streptococcus viridans fæcalis* with streptomycin. That the drug can be used effectively in this type of infection is illustrated by the case report of Guss (1948).

Clinical Record.

H.B., a man, aged sixty-one years, was admitted to hospital on June 6, 1948, complaining of weakness, lassitude and loss of weight (two stone) over a period of two years. He had enjoyed good health in the past and the onset of his present illness had been very insidious. On specific interrogation he stated that he had noticed breathlessness on exertion for about three years and anorexia for six to nine months. His presenting symptoms of weakness and lassitude had been intensified in the three months immediately prior to admission, and he had had rigors and felt feverish during this period. Review of other symptoms, past history and family history provided no relevant information.

Physical examination revealed a thin, pale, elderly man, lying quietly in bed and appearing seriously and chronically ill. The skin was dry and clear. The tips of the fingers were tender, but no petechial hæmorrhages or embolic nodes were observed. There was no clubbing of the fingers. The nose and throat were normal and the examination of the mouth revealed no significant abnormality, there being full artificial dentures. Pallor of the skin and mucous membranes was well marked. There was no palpable enlargement of the lymph glands.

The chest expanded equally and symmetrically. The percussion note was resonant throughout, the breath sounds were vesicular and there were no accompaniments.

The pulse was regular and normal in other respects, but the vessel wall was palpable. The blood pressure was 165 millimetres of mercury (systolic) and 100 millimetres (diastolic). The apex beat was visible and palpable in the fifth intercostal space four and a half inches from the mid-sternal line. A harsh organic systolic murmur was audible at all areas but loudest at the mitral area. There were no precordial impulses or thrills.

The abdomen was soft and scaphoid. There was moderate epigastric tenderness. The liver was palpable one finger's breadth below the right costal margin, but was not tender. The spleen was palpable two fingers' breadth below the left costal margin, but was not tender.

Examination of the central nervous system revealed no abnormality. The ocular fundi were normal.

Repeated examination of the urine showed an average of 10 to 12 blood cells and several granular casts per high-power field.

Examination of the blood showed a hæmoglobin content of 12 grammes *per centum* and a total per cubic millimetre of 3,400,000 erythrocytes and 5000 leucocytes; of the latter 73% were neutrophile cells, 25% lymphocytes and 2% monocytes.

Culture of the blood on five separate occasions consistently yielded a growth of a streptococcus, the report on which read as follows:

> Sub-culture after 5 days' incubation grows a streptococcus with an indistinct production of L-Hæmolysis on blood agar. The colony is large (2 mm. in 48 Hrs.) and the Streptococcus is in short chains. There was no fermentation of Mannite nor will it grow in the presence of bile salts. This is a streptococcus probably of the viridans group of unusual type. Growth is inhibited by Penicillin down to a concentration of 0-15 units/ml.

Radiological examination of the heart revealed slight generalized enlargement of the cardiac shadow. The lung fields were normal except for some old tuberculous scarring at the right apex. There was no evidence of any active infection. The electrocardiogram showed no significant change.

The patient had come under my care on August 28, 1948, having been transferred from another hospital, where the diagnosis had been made and where he had been treated with penicillin. He had been given a total of 32,000,000 units, at first in dosages of 100,000 units four-hourly, and in dosages of 250,000 units from July 12 to July 25, 1948, when the course was completed. As the patient had become pyrexial again on July 27 and the result of blood culture on a fourth occasion proved to be positive, a further course of penicillin in doses of 250,000 units four-hourly was begun on August 28. We continued this therapy in the same dosage until August 31, when the dosage was increased to 500,000 units four-hourly.

Penicillin administration was suspended on September 9, when 30,000,000 units had been given in the second course with a total of 65,000,000 units of penicillin in the two courses. The patient's condition had not improved, and the result of blood culture on September 12 was still positive.

The patient's condition continued to deteriorate. He was restless, febrile and delirious part of the time, and on September 20 death seemed imminent. On this date treatment was begun with streptomycin, three grammes daily being given in divided doses at four-hourly intervals.

On October 1 the patient's condition was much improved, the temperature had returned to normal, and he felt better in every way. The blood became consistently sterile. On October 26 the patient noticed slight subjective vertigo and the dosage of streptomycin was reduced to two grammes daily. Ascorbic acid (100 milligrammes) and "Polyhæmin" (one tablet) were administered three times a day.

On November 1 streptomycin therapy was discontinued, a total of 90 grammes having been given. The patient was discharged from hospital ten days later, feeling well except for weakness and slight subjective vertigo.

Now, six months later, the patient has almost regained his normal weight (weight increase one and a half stone since discharge) and feels well except for vertigo. The spleen is no longer palpable. The blood has remained sterile, the sedimentation rate has returned to normal, and the hypochromic normocytic anæmia present on discharge from hospital is cured. There is no evidence of congestive cardiac failure, although there is moderate left ventricular enlargement and an organic systolic murmur present at the mitral area.

Summary.

A case of subacute bacterial endocarditis due to *Streptococcus viridans* of unusual type is presented.

It is considered to be of special interest because of the apparent response to streptomycin after penicillin in

huge dosage had failed despite moderate sensitivity to penicillin *in vitro*.

Recognition has been given to the fact that spontaneous remissions occur in this disease, but the condition of the patient was so low and the improvement within a week of the institution of streptomycin therapy so remarkable that it is felt that streptomycin undoubtedly brought about the remission.

Cure, of course, is not yet claimed; but it is felt that this case demonstrates that streptomycin may be of value in those cases of subacute bacterial endocarditis in which penicillin in adequate dosage has failed after due trial, particularly if the causative organism is a *Streptococcus viridans* of unusual type.

Acknowledgement.

I wish to thank Dr. John Garvan and the staff of the Pathology Department, Lewisham Hospital, who carried out all pathology investigations in this case.

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DETACHMENT OF THE RETINA: A DESPERATE CASE.

By ARTHUR D'OMBRAIN, M.B., Ch.M., D.O.M.S., F.R.A.C.S. (Oph.),
Sydney.

THERE occur in the experience of us all, cases that force the choice of saying either that we feel the patient's condition is hopeless or that drastic measures, involving grave surgical risks, offer a slight chance of success.

Clinical Record.

The patient, Miss L.M.W., aged thirty-five years, was slightly deficient mentally. She lived with her parents in a country district, where her chief recreation was riding, at which she was expert. About fifteen months ago, after returning home from an afternoon's ride, she suddenly noticed a dark cloud in front of the left eye, which subsequently lost all vision except perception of light. She had always been short-sighted, but as the right eye still had adequate distance sight, with the aid of glasses, and as she could still read without glasses, no doctor was consulted.

About four months ago, again when she was returning from a ride on horseback, several thick, grey-coloured, wavy lines appeared in the upper part of the visual field of the right eye. Within two days the lines coalesced into a long, horizontal, oval, dark shadow.

By the time her parents had been able to make an appointment with an oculist in the nearest country city, central vision had become obscured. The oculist diagnosed retinal detachment, which he considered hopeless in both eyes.

Her distracted parents then took her to a Sydney oculist, who told them that the condition of their daughter's left eye was hopeless and that of her right eye almost hopeless. He advised them, however, to allow him to try to reattach the retina in the right eye, and he performed a scleral-surface coagulation diathermy operation with immediate good results. On the sixth day after the operation the lesion relapsed, with loss of central vision.

A fortnight later her parents brought her to me. I telephoned my colleague, who most generously gave me the whole history and wished me the best of fortune in anything I might consider trying in this, in his opinion, a hopeless case.

On examination I found the left eye to have a total retinal detachment, vision being barely perception of light. The right eye, which could recognize hand movements at a distance of two feet, was found to have a complete, balloon-like detachment of the lower part of the retina, with adhesion traction lines extending upwards to the *macula lutea*. There was no hole in the retina to be discerned, nor had the surgeon who first operated upon her found any retinal hole.

At first I was tempted to take the easy way out, but the distress of the parents and also of the patient, who was not so unintelligent that she could not understand that physical darkness would soon be added to her mental gloom, influenced me in deciding to make a final attempt.

Having made this decision, I determined that all the usual considerations of how much damage the retina or vitreous should be called upon to bear would have to be cast aside, as nothing short of the most drastic measures would have any chance of success, in view of the failure of an efficiently performed orthodox operation.

Ordinarily, in an operation for the surgical relief of separation of the retinal layers, even though perforating diathermy may have to be added to surface scleral coagulation, every care is taken to avoid penetration of the retina itself, owing to the risk of causing vitreous disturbance, with resultant cloudiness of the vitreous medium and the formation of the fibrous bands known as *retinitis proliferans*, which may themselves draw away some of the retinal layers. In this case this risk had to be taken. Also it is usual not to perforate the sclera and chorioid until the completion of scleral-surface diathermy, owing to the disadvantage of operating upon a soft and semi-collapsed ocular globe. Again, then, in this case it was judged advisable to evacuate the subretinal fluid at the beginning of the operation, so that the retina would collapse outwards against the chorioid and lie within reach of the diathermy needles.

Accordingly, with the patient under "Pentothal" anæsthesia, expertly conducted over a period of two and a half hours by Dr. Stuart Marshall, an operation was performed, with use of the Schoenberg two-pronged curved hook and the combined cutting and coagulation high-frequency current, devised by the Liebel-Flarsheim Company of Cincinnati, Ohio, United States of America, and originally brought to my notice by Mr. Hunt and Mr. Wilson, of the Stanford X-Ray Company, Sydney.

On reflection of the conjunctiva and severing of the right inferior rectus muscle, the scleral scars, eight in number, left by the previous surface diathermy operation were clearly to be seen. The Junior-Bovie diathermy unit was switched to the scalpel or cutting current and the Liebel-Flarsheim current modifier was plugged into the machine. This modifier converts the scalpel or cutting current into a half-cutting, half-coagulation current.

The electrode used for penetration of the sclera was the Schoenberg double-pronged hook, and with this instrument 15 perforations of the sclera were made, covering the entire area between the external and internal rectus muscles, nine of them as far posteriorly as possible and six of them more anteriorly. The first perforation, which was placed most posteriorly of all, released the subretinal fluid and the eyeball became flaccid; but this did not matter, as the Schoenberg hook acts as a small retractor, and no difficulty was experienced in making the rest of the punctures.

At the first puncture, therefore, the release of the subretinal fluid allowed the separated retinal layers to collapse against the chorioid and, as the Schoenberg hook is long enough to penetrate the retina, if plunged right in, subsequent punctures perforated the retina. This is bad surgery in the usual case of detached retinal layers, but this was not a usual case; how unusual it was, I did not realize until the fourth day after operation, when the patient developed an attack of acute mania.

It is desirable after an operation for detachment of the retina to keep the patient and the patient's eyes as still as possible for several weeks. In this case, from the fourth day after operation such a procedure became impossible of attainment. From a pleasant-natured, semi-moronic type the patient became a violent lunatic, requiring four people to hold her down. Sedatives, including "Nembutal", paraldehyde, bromides and morphine, had no effect. The bandages were removed from her eyes, all eye treatment was forgotten, and admission to the Reception House was rendered imperative. From there two days later she was admitted to a mental hospital, where she gradually recovered from her mania and eventually reached the mental level which for her was normal.

On examination of the patient at my surgery about a month after her operation all the previously detached retina was reattached, the discrete coagulation areas, about one-quarter to one-half optic disk diameter in size, being clearly seen. Visual acuity for distant vision, about $^{3}/_{18}$ with a *minus* eight lens, was not marvellous, except in view of the particular circumstances; that for near vision was J3, which means that she could read ordinary newstype, and she read fluently, and with great pride and delight, a letter written to her by her mother while she was in the mental hospital.

There were a few fine vitreous "floaters" to be seen, it is true, but what were these compared with the restoration of useful visual functions to an unfortunate human being, whose only residual pleasure was the perception of the features of those whom she loved?

Summary and Conclusions.

A case has been described of a woman of deficient mentality with one blind eye, who had an extensive retinal detachment of the other eye, previously operated upon by a competent ophthalmic surgeon by the surface diathermy technique. It would appear that in desperate cases of this nature an extensive and deep penetration of the coats of the eyeball by a combined cutting and coagulation high-frequency diathermy current offers a chance of success.

Reviews.

A GENERAL HISTORIAN EXPLORES THE MEDICAL FIELD.

It is exceptional for a general historian to find the time or to indulge an inclination to delve deeply into the depths of that vast medical literature which has accumulated over the past centuries and to become conversant with the manifold works of those authorities who have carried out original research in the subject. This unusual vocation has been fully sustained by Dr. Richard Harrison Shryock, who occupies a dual position as Professor of American History and Lecturer in Medical History at the University of Pennsylvania. For some time Professor Shryock has been recognized as an authoritative writer on historical subjects, and now his book, "The Development of Modern Medicine", has been reprinted for the third time since 1936.[1]

There is no doubt that Dr. Shryock has been able to superimpose upon his basic knowledge of general history an extensive study of many phases in the development of medicine and the allied sciences; and he brings to bear all the latest technique of modern historical methods in the presentation of this book. Nearly every page and every statement is copiously documented by references to a mass of miscellaneous literature from Herodotus to the *New York Times*. Perhaps we feel rather envious that so much material is available to the research scholar in that favoured region of the United States of America; but to a hardened product of the old school it is at times tedious and bewildering in an endeavour to come to terms with the author to sort out the evidence and follow the trend of his ideas.

Some helpful remarks may be found in the preface to encourage the reader who is capable of realizing that it is harder to dig for gold than for potatoes; for here the author explains that "This book is in no sense a study in the technical history of medicine, but is rather an attempt to portray certain major aspects of medical development against a background of intellectual and social history in general. Viewed in this perspective, the progress of medicine may take on a new form and meaning". Few medical historians could quibble with such a contention. Nevertheless it behoves every writer seeking to present a new viewpoint to exercise special care in the presentation of his work, and to see that his comments and interpretations of the evidence are set out with crystal clarity. The publisher of this book states on the dust cover that it is one for the medical man, research chemist, biologist, psychologist, social worker and the layman. Well, that is just another of its problems that we feel unable to solve.

ELEMENTARY ANATOMY AND PHYSIOLOGY.

"ELEMENTARY ANATOMY AND PHYSIOLOGY", by Professor James Whillis, of Guy's Hospital Medical School, has reached a third edition, thus indicating that this work is serving the purpose of the author to meet the needs of the elementary student who is likely to be bewildered by the fuller text-books of anatomy.[1]

Systematic anatomy occupies the greater part of the book's nineteen chapters, the remainder being devoted to "some simple physiology", including sections dealing with digestion, the blood, metabolism, the ductless glands, and the spleen and liver.

It is not possible to agree with Professor T. B. Johnston that the author has succeeded in correlating structure and function to any great extent, although occasionally he has done so, particularly in his account of posture, and in his analysis of walking, and ascent and descent of stairs. These matters, incidentally, cannot be regarded as elementary. On the other hand, the account of individual muscles is in some instances very elementary, and in fact is set down so briefly as to resemble a catalogue.

The description of the circulation in the arteries and arterioles does not appear to be in accord with the usual teaching, and, generally speaking, the elementary physiology is not up to the standard of some of the well-known books on this subject. Perhaps it is not possible to combine successfully in the one volume elementary anatomy and elementary physiology. An outline of the latter utilizes only as much anatomy as is necessary, but up to the present we have not seen a book on elementary anatomy that makes use of as much physiology as is necessary.

The book is of convenient size, well printed, and clearly illustrated, although occasionally the diagrams present inaccuracies, which is unfortunate, even in an elementary text-book.

ORTHOPTICS.

MARY EVEREST KRAMER'S "Clinical Orthoptics Diagnosis and Treatment" is designed as a text-book for orthoptic students with the secondary purpose of giving the ophthalmologist a "working knowledge" of the subject.[2]

In this second object the book succeeds admirably. It contains a very clear exposition of the details of instruments for diagnosis and treatment and of the methods of their use. There is also much sound common sense in references to the handling of young patients and their parents, given by an obviously experienced orthoptist.

The author has made no attempt to go beyond the orthoptist's sphere, and consequently the surgeon will find little consideration of the relationship of surgery to orthoptics or indication of the factors to be considered in timing an operation. The rather bare bones of anatomy, physiology and optics are there, and the essentials of practical aspects of strabismus, shorn of most theoretical considerations.

[1] "The Development of Modern Medicine: An Interpretation of the Social and Scientific Factors Involved", by Richard Harrison Shryock; 1948. London: Victor Gollancz, Limited. 8½" × 5½", pp. 388. Price: 21s.

[1] "Elementary Anatomy and Physiology", by James Whillis, M.D., M.S., F.R.C.S., with a foreword by T. B. Johnston, M.D., Ch.B.; Third Edition; 1949. London: J. and A. Churchill, Limited. 9¼" × 6¼", pp. 308, with 108 illustrations. Price: 16s.

[2] "Clinical Orthoptics Diagnosis and Treatment", by Mary Everist Kramer, edited by Ernest A. W. Sheppard, M.D., and Louisa Wells-Kramer; 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¾" × 6½", pp. 480, with 147 illustrations. Price: 60s.

There is no reference to Travers's work on concomitant strabismus, and prognosis is considered only in the broadest sense and little aid is given to the student in forecasting the time that treatment is likely to take for each type of case. A surprising omission is the lack of mention of contraindications to occlusion, such as nystagmus.

It is clear that the author has a sense of vocation to her art and is a sincere and ethical advocate for orthoptics, even down to frank and detailed personal advice to orthoptists. However, though lucid and admirably produced, with good print and clear illustrations, the book is too incomplete to be called a reference book.

ELEMENTARY BOTANY.

"INTRODUCTORY BOTANY", by Alexander Nelson, provides the first year university student and the matriculation student with a valuable addition to his botanical library.[1] It may be used as a text-book or as a book of reference. The matter covered is wide, and examples are well and carefully selected.

There is no pretence that this book should be a guide to practical work. Certain experiments are given and described in the physiology section, but the author points out in the preface: "It has been assumed that each student will have access to such botanical specimens and experiments as may be necessary to illustrate the various parts of the subject. So, too, it is hoped he will draw and describe what he sees in these exhibits and so become accustomed to providing an illustrated discussion of an organism, its parts, or its function." Thereby he stresses what is so important in early scientific studies, that the student should learn to observe and to record accurately all that he sees and does.

The text is well illustrated throughout with excellent line diagrams. The block diagrams in the morphology section are most instructive. The clear diagrams and tables in the sections dealing with the types give a very simple explanation of alternation of generations in each case, together with a clear and concise method of learning these life-histories.

The student using this text-book will realize that in order to understand thoroughly the biological sciences, it is necessary to have a fundamental training in the physical sciences.

FORENSIC MEDICINE.

THE fifth edition of "Mollison's Forensic Medicine Lectures" has been revised and enlarged by Dr. Keith Bowden, of the University of Melbourne.[2] This book approaches the subject in a very practical manner, presenting the reader with the essentials of forensic medicine and avoiding much of the detail and technical procedure found in larger books on the subject.

The opening chapter gives a comprehensive survey of the conditions, covered by the term natural causes, which may cause sudden or unexplained death. This is followed by a description of the changes which take place in the body after death and a concise chapter on the various points concerned in identification. Injuries, bullet wounds, the various forms of asphyxial death, abortion and the newborn child are dealt with in the customary manner. There is a good chapter on the often neglected simple subjects of deaths from burns, electricity, heat, cold and shock. The chapters on toxicology give the law in regard to poisons, and then the main poisons are set out in a manner which should appeal to students. A little more might have been written in some instances, for example, on the barbituric acid group in view of the increasing importance of these drugs. The closing chapters will give the student, and even the general practitioner, some excellent advice on various medico-legal problems, the performance of the autopsy, the giving of evidence in the law courts, and their responsibilities in legal medicine.

The book is primarily one for Victorian students and should be eminently suitable for them.

[1] "Introductory Botany", by Alexander Nelson, B.Sc. (Glasgow), Ph.D., D.Sc. (Edinburgh); 1949. Edinburgh: E. and S. Livingstone, Limited. 8¼" × 5¼", pp. 492, with 121 illustrations. Price: 22s. 6d.

[2] "Mollison's Forensic Medicine Lectures", Revised, Enlarged and Illustrated by Keith Macrae Bowden, M.S.; Fifth Edition; 1949. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 8½" × 5½", pp. 296, with 65 illustrations. Price: 37s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The 1948 Year Book of Neurology, Psychiatry and Neurosurgery"; Neurology, edited by Hans H. Reese, M.D., and Mabel G. Masten, M.D.; Psychiatery, edited by Nolan D. C. Lewis, M.D.; Neurosurgery, edited by Percival Bailey, M.D.; 1949. Chicago: The Year Book Publishers. 7" × 4½", pp. 754, with 140 illustrations. Price: $5.00.

One of the "Practical Medicine Series" of year books.

"Principles of Health Education Applied", by Clifford Lee Brownell, Ph.D.; 1949. New York, Toronto, London: McGraw-Hill Book Company, Incorporated. 9" × 5½", pp. 388, with 16 illustrations. Price: $3.75.

Intended for use by students of health education in schools and colleges.

"Fundamentals of Internal Medicine", by Wallace Mason Yater, A.B., M.D., M.S. (in Med.), F.A.C.P.; Third Edition; 1949. New York: Appleton-Century-Crofts, Incorporated. 9¾" × 6¼", pp. 315. Price: $12.00.

Designed "to make readily available in simple form for student and practitioner the essentials . . . of internal medicine". There are nineteen contributors.

"The Mental Life of the Child", by Gustav Hans Graber; 1949. London: Staples Press, Limited. New York: Staples Press Incorporated. 7" × 5¾", pp. 164. Price: 8s. 6d.

Addressed to all preceptors, parents, teachers, priests, physicians, psychologists and students.

"A General Survey of the British Pharmacopœia, 1948"; a series of lectures by members of the British Pharmacopœia Commission, 1933-1948, and other authorities; 1949. London: The Pharmaceutical Press. 8¼" × 5¼", pp. 88. Price: 5s. 6d.

Intended for students and pharmacists.

"The Epidemiology of Hemolytic Streptococcus: During World War II in the United States Navy", by Alvin F. Coburn, M.D., and Donold C. Young, M.D.; 1949. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson. 9" × 5¾", pp. 242. Price: 30s.

Concerned with happenings in the United States Navy as they occurred and with attempts to interpret their interrelation. No attempt is made to review the literature.

"Anæsthetics for Medical Students", by Gordon Ostlere, M.A., M.B., B.Chir. (Camb.), D.A., with a foreword by C. Langton Hewer, M.B., B.S., M.R.C.P., F.F.A.R.C.S.; 1949. London: J. and A. Churchill, Limited. 7¼" × 4¾", pp. 116. Price: 7s. 6d.

An attempt to put all the essential data for safe and satisfactory anæsthesia in as short and informal a form as possible.

"The Physique of Young Adult Males", by W. J. Martin; Privy Council, Medical Research Council Memorandum, Number 20; 1949. London: His Majesty's Stationery Office. 9¼" × 6", pp. 72. Price: 1s. 3d.

Based on the study of data obtained from the medical examination of 90,000 young men between the ages of twenty and twenty-one years.

"Cardiovascular Disease: Fundamentals, Differential Diagnosis, Prognosis and Treatment", by Louis H. Sigler, M.D., F.A.C.P.; 1949. New York: Grune and Stratton Incorporated. 9" × 6", pp. 574, with many illustrations. Price: $10.00.

The author has attempted to cover all the problems which the practitioner is called upon to solve in his practice.

"Regional Ileitis", by Burrill B. Crohn, M.D.; 1949. New York: Grune and Stratton Incorporated. 8¼" × 5¼", pp. 238, with many illustrations.

A comprehensive study of the subject with 257 references to the literature.

"Shaw on Vivisection", compiled and edited by G. H. Bowker for the National Anti-Vivisection Society; 1949. London: George Allen and Unwin, Limited. 7¼" × 5", pp. 66. Price: 5s.

Shaw's views on animal experimentation have been revised by him for republication by the National Anti-Vivisection Society.

"A History of Oto-Laryngology", by R. Scott Stevenson, M.D., F.R.C.S. (Edinburgh), and Douglas Guthrie, M.D., F.R.C.S. (Edinburgh); 1949. Edinburgh: E. and S. Livingstone, Limited. 8¼" × 5¼", pp. 162, with many illustrations. Price: 17s. 6d.

Describes the rise and progress of the specialty from the earliest times to the present.

The Medical Journal of Australia

SATURDAY, AUGUST 20, 1949.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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DRUG ADDICTION: HEROIN AND THE BARBITURATES.

THE World Medical Association has recently interested itself in the problem of habit-forming drugs. It has done this as a result of a request for collaboration from the World Health Organization. The World Medical Association has written to each of its member national medical organizations asking for its opinion and points of view. First of all the World Health Organization wishes to secure information on the use and dispensability of diacetylmorphine (heroin) and an estimate of the amount used in each country. Secondly, it is interested in the establishment of a mechanism for giving a single name to every habit-forming drug subject to international control. This matter was mentioned at the recent meeting of the Federal Council of the British Medical Association in Australia. The Federal Council resolved to obtain what information it could in regard to the first of these two aspects and to forward it to the World Medical Association. It would appear that what was really wanted was some opinion on the dispensability of heroin; the amount of opium-containing drugs used in the Commonwealth could easily be discovered by the World Health Organization because each country must state every year the amount of these drugs required by it. This annual budget is sent to the Permanent Opium Advisory Committee of the United Nations Organization. The manufacture and distribution of narcotic drugs were the subject of a convention called by the Assembly of the League of Nations in 1931. The League described the convention as "a bold conception without precedent in the history of international relations and international law". As a matter of fact it was preceded by The Hague Convention of 1912 and the Geneva Convention of 1925. It was at The Hague Convention that the contracting powers agreed to apply laws and regulations for the manufacture, importation, sale or exportation of morphine, cocaine and their respective salts. This agreement applied (*a*) to medicinal opium, (*b*) to all preparations (official and non-official, including the so-called anti-opium remedies) containing more than 0·2% of morphine or more than 1% of cocaine, (*c*) to heroin, its salts and preparations containing more than 0·1% of heroin, (*d*) to every new derivative of morphine, cocaine or their respective salts or to any other alkaloid of opium which might after generally recognized scientific investigations give rise to similar abuse and result in the same injurious effects.[1] The use of narcotic drugs in Australia is controlled by State Acts. In New South Wales alone is the policing of the regulations under the Act in the hands of the Police Department and the control exercised is very efficient. In most instances addiction to heroin arises as a result of transference from morphine. In New South Wales there are possibly half a dozen heroin addicts, but the number of persons addicted to morphine is very much greater. Heroin is more powerful than morphine; in consequence addiction to it is more easily acquired and is more serious than addiction to morphine. Australia is among the highest *per capita* consumers of heroin in the world—it is grouped in this regard with Finland, Italy, New Zealand and Sweden. If the number of addicts in New South Wales is representative of the numbers in the other Australian States, we must conclude that the therapeutic use of the drug is wisely controlled by the medical profession. In these circumstances the practising members of the profession in this country will hesitate to say that they can dispense with heroin. The proposal that the manufacture of heroin should be abandoned has been made more than once. The League of Nations Convention in 1931 recommended that the several governments should examine the possibility of prohibition of manufacture. Terry and Pellens quote similar recommendations by several organizations and by individual investigators, some of them as long ago as 1918.

It will be more appropriate and also more profitable to refer to other habit-forming drugs as well as to heroin. The "present status of narcotic addiction" has recently been discussed by V. H. Vogel, H. Isbell and K. W. Chapman.[2] These authors write from the United States Public Health Service Hospital, Lexington, Kentucky, and they read their paper at the annual meeting of the American Medical Association in June, 1948. They define drug addiction as a state in which a person has lost the power of self-control with reference to a drug and abuses the drug to such an extent that the person or society is harmed. They refer to a description by C. K. Himmelsbach and L. F. Small of addiction to opium and similar drugs as embracing three intimately related but distinct phenomena—tolerance, physical dependence and habituation. These phenomena are interesting and form a basis for the discussion of other drugs. It should, however, be noted that many divergent opinions have been expressed on the nature of addiction. Terry and Pellens cover many pages with the views of authors. C. W. Carter, for example, wrote in 1908 that morphinism was not a disease or an immorality, but a derangement of function, a change in the mode of action of the organism, not a change in its structure. It was, he thought, a condition of auto-intoxication for the relief of whose discomforts morphine was taken, a condition of double poisoning, first and least by the drug and mostly by the toxins of the body itself. Oscar Jennings in 1909 stated that the morphine "habit" was a "psychosomatic affection" in which the mental and

[1] Quoted by C. E. Terry and M. Pellens in "The Opium Problem", published by The Committee on Drug Addictions in collaboration with the Bureau of Social Hygiene, Inc., New York, 1928.

[2] *The Journal of the American Medical Association*, December 4, 1948.

physical troubles were interdependent, conditioned the one by the other. C. L. Case in 1910 held that morphinism was a disease as really as typhoid fever or pneumonia was a disease; the "craving" was not a craving at all, but a physical condition. Daniel Morat in 1911 stated that, regardless of the cause of the condition, morphinism was a true organic disease which had to be treated like any other chronic intoxication, moral treatment being only to prevent relapse. Foster Kennedy in 1914 said that morphinism was a disease, in the majority of cases initiated, sustained and left uncured by members of the medical profession. In 1920 the Council on Health and Public Instruction of the American Medical Association issued a report in which it referred to those who used narcotic drugs for the comfort they afforded and continued their use solely by reason of an acquired habit. It regarded these persons as suffering from a functional disturbance with no physical basis expressed in pathological change. In 1926 the Departmental Committee on Morphine and Heroin Addiction of the Ministry of Health of Great Britain issued a report in which the following definition of an addict was given: "A person who not requiring the continued use of a drug for the relief of the symptoms of organic disease, has acquired as a result of repeated administration, an overpowering desire for its continuance and in whom withdrawal of the drug leads to definite symptoms of mental or physical distress or disorder." The committee stated that there was general agreement among its witnesses that in most well-established cases the condition of addiction had to be regarded as a manifestation of disease and not as a mere form of vicious indulgence.

Having noted these varying opinions on the nature of morphine addiction, we may return to the three phenomena mentioned by Himmelsbach and Small—tolerance, physical dependence and habituation. Tolerance is defined as "a diminishing effect on repetition of the same dose of the drug or, conversely, a necessity to increase the dose to obtain an effect equivalent to the original dose when the drug is administered repeatedly over a period of time". Physical dependence refers to "an altered physiologic state, brought about by the repeated administration of a drug over a long period of time, which necessitates the continued use of the drug to prevent the appearance of the characteristic illness which is termed an abstinence syndrome". Habituation refers to "emotional or psychologic dependence on the drug—the substitution of the drug for other types of adaptive behaviour". Vogell, Isbell and Chapman point out that the importance of these three aspects in the total picture of addiction is still a matter of dispute. Pharmacologists, particularly, regard physical dependence as the primary and only distinguishing characteristic product of an addicting drug. Others, particularly psychiatrists, look on emotional dependence or habituation as the all-important factor in addiction. The point to which we wish to draw attention about these three criteria, which obviously apply to morphine and heroin, is that they also apply to the barbiturates, and the barbiturates cause in Australia more deaths and what are known as near-deaths than any other group of drugs.

The key to the prevention of addiction in most instances lies in the statement of Foster Kennedy, already quoted, that morphinism is in the majority of cases initiated, sustained and left uncured by members of the medical profession. If we substitute "drug addiction" for morphinism and, much as we may dislike the statement, base our actions on it, we shall go a long way towards effecting a remedy. In this matter a start must be made by the early indoctrination of the student. It is said of a well-known Australian clinical teacher and examiner of an early generation that he would unhesitatingly fail a student in his "finals" if the first remedy he offered for the relief of a patient's pain was the exhibition of morphine. One of Australia's leading psychiatrists holds that many of the present-day graduates of Australian medical schools are not sufficiently mindful of the safeguards that should attend the ordering of habit-forming drugs. This is something that clinical teachers will do well to consider. It may possibly be an accusation against their teaching; if it is, the point should be critically examined and not resented. The occasions on which the taking of a habit-forming drug may be left to the patient's decision are very rare. For practical purposes it would be better to regard them as non-existent. The old saying that prevention is better than cure may in regard to habit-forming drugs be altered to read that prevention is easier than cure.

Current Comment.

THE LABILE ELEMENT OF HYPERTENSION.

IN the everyday procedure of estimating the diastolic and systolic blood pressure it is sometimes important to know what degree of variation is caused by emotional or other factors. J. L. Frew and M. L. Rosenheim, in comparing the results of blood pressure estimations after the administration of tetra-ethyl-ammonium bromide and a rapidly acting barbiturate, remind the reader that the basal blood pressure should be determined under conditions similar to those used for the estimation of the basal metabolic rate.[1] This was a recommendation of a joint committee of the Cardiac Society of Great Britain and the American Heart Association in 1939. The experienced physician can no doubt secure an approximately correct reading if he insists on maintaining the ideal of a leisurely approach, but ideals fall from their own pedestal as life becomes more crowded, and as the physician's own blood pressure rises. It will now be readily accepted that an accurate diastolic blood pressure reading is essential in order to know the degree of constant strain on the circulation. Lability is a notorious quality of blood pressure levels, and though the range is less in diastolic readings, it is important. Frew and Rosenheim investigated 41 patients, mostly suffering from benign essential hypertension. They estimated the initial blood pressure, which is, of course, subject to many fluctuations, and then, with the patient resting under basal conditions, the resting blood pressure. The particular interest in this inquiry lay in the comparison of the latter reading with those obtained after the administration of tetra-ethyl-ammonium bromide and "Seconal" respectively. The former drug was administered intravenously in solution, the usual dose being as a rule 500 milligrammes. Lower doses were given to small or feeble patients, and larger doses were unnecessary. Only one disturbing reaction occurred, and it was readily abolished with the use of adrenaline. The minimum reading of the blood pressure was determined by making observations every half-minute, and was reached as a rule in five minutes or less. The effect was transitory only. At a separate session "Seconal", chosen by reason of its rapid action, was given to the patient when he was lying comfortably in his own bed in a quiet room. The blood pressure

[1] *Clinical Science*, Volume VII, 1949, Numbers 3 and 4.

was observed every five minutes till the minimum reading was reached. As a rule deep sleep was produced by a dose of four and a half grains, or less in frail patients, but some required six grains. The average fall in the diastolic pressure caused by tetra-ethyl-ammonium bromide was greatest in subjects whose initial diastolic pressure was between 110 and 129 millimetres of mercury, and least in those whose initial readings were between 90 and 109 millimetres. It was found that a sharp but usually transitory rise followed the insertion of a needle in a vein in all patients, which is not surprising. Sometimes this emotional rise persisted. "Seconal" usually produced sleep within fifteen to thirty minutes, and though the patient could as a rule be easily roused, a minimum reading could easily be obtained without disturbance. The minimum readings for the diastolic pressure obtained by each of these methods was substantially the same. Tetra-ethyl-ammonium bromide is believed to act on the autonomic ganglia, and "Seconal" at a rather higher level, but in any case the authors suggest that their action is on the neurogenic element of hypertension. Using this technique, they have found that patients suffering from hypertension in a malignant phase still show a high diastolic pressure. The neurogenic element appears to be of little significance in the hypertension of acute glomerular nephritis, as might be expected. Frew and Rosenheim suggest that the basal blood pressure results from a number of factors, chiefly the organic structural state of the arterioles, intrinsic vascular tone of humoral or other origin, and the volume of circulating blood. What they term the "casual" blood pressure, meaning a chance or initial reading, is affected also by superadded neurogenic tone. They conclude that the methods they have employed provide simple and reliable methods of estimating the minimum diastolic blood pressure.

PERIARTERITIS NODOSA AND THE SULPHONAMIDES.

THE growing frequency of references to *periarteritis nodosa* in the medical literature has been remarked by a number of writers. Evidence favouring an hypothesis that this disease complex is due in part at least to drug sensitivity has been brought forward by a number of authors, and, while the position is far from clear as yet, it would seem possible that the beneficence of the sulphonamide drugs may be tempered somewhat by yet another toxic property. Perhaps it is curious that so many medical investigators appear to be very interested in this disease complex, which has inspired an increasing number of articles in medical literature of recent years. Maxwell L. Gelfand and Solomon Aronoff have recently collected some figures and information from the records of the Bellevue Hospital in New York, and present these in the form of a review of two periods, 1916 to 1937, and 1938 to 1946.[1] Four cases of *periarteritis nodosa* were recorded in the former period and fourteen in the latter. In the latter series the clinical diagnosis was proved by either biopsy or necropsy. It is admitted, of course, that a symptom complex which involves the vascular tree must be expected to present clinical puzzles. The originally described symptoms and signs have been considerably extended of recent years, and at the present time these are usually taken to include groups of symptoms referable to sepsis of some non-obvious origin, those arising from involvement of the arterial systems of various organs, allergic manifestations affecting the blood, the arteries themselves, and maybe the respiratory system, and demonstrable nodules discovered by touch or microscopic section of skin or muscle. In the Bellevue series the clinical findings included a history of asthma, polyneuritis and polymyositis, fever and cachexia, retinal lesions, nephritis, and lesions in the skin and the joints. The blood changes included anæmia, leucocytosis and eosinophilia. The diagnosis was clinched by biopsy of muscle or skin. It is of interest that in three instances a positive result to a test for trichina infection was obtained. The pathological pattern found in the biopsies was consistent with that recognized in this disease complex, and included a necrotic inflammatory phase, with alteration of the media of affected arteries, and perivascular infiltration with cells that usually included eosinophiles. Resolution may lead to the formation of firm organized nodules, but progressive lesions of the intimal coat of the vessels may cause thrombosis, and with involvement of the medial coat, aneurysms may form. It was found that males in this series predominated over females by almost two to one, which accords with the findings of others. A high proportion of the patients had an associated history of asthma, and eosinophilia occurred in nearly half the patients. The connexion between these findings and the exhibition of sulphonamides in this series is only by assumption, though the authors' arguments are suggestive of some connexion. They quote the well-known work of Rich on the subject, in both the clinical and the experimental spheres, which strongly supports the theory that *periarteritis nodosa* is a manifestation of hypersensitivity.

Gelfand and Aronoff admit that increase in recorded cases may well be due in part to greater knowledge and interest; but they also point out that analysis of autopsy material at the Johns Hopkins Hospital by Rich showed that a steady increase in numbers of recognized cases of this condition has taken place during the years in which sulphonamide treatment has become more common, or indeed practically universal. The possibilities of sensitization of members of the public by taking some of the magic tablets successfully used by a friend can only be guessed. The authors quote work showing how sensitivity to sulphonamides occurs, and the possibilities of forming a drug-plasma protein complex which can act as an antigen. It is known, too, that conjugated sulphonamide compounds can produce anaphylaxis in animals, and more convincing is the now well authenticated evidence of vascular changes resembling *periarteritis nodosa* appearing in the human subjects of sulphonamide administration. The present authors claim that inasmuch as vascular lesions resembling *periarteritis nodosa* have been described with greater frequency in patients and experimental animals receiving these drugs, it is reasonable to assume that there is a causal relation.

[1] *Annals of Internal Medicine*, May, 1949.

THE KARP STRAIN OF RICKETTSIA TSUTSUGAMUSHI.

DURING the recent war a strain of *Rickettsia tsutsugamushi* was isolated in Brisbane which has since been used extensively in experimental work on scrub typhus. It is known as the Karp strain, as it was isolated from the blood of an American soldier named Karp. Because of conflicting accounts of the origin of the strain, E. H. Derrick and H. E. Brown[1] have recorded the circumstances of its isolation. Karp was wounded while in the Buna-Gona area of New Guinea and evacuated to the 42nd United States Army Hospital in Brisbane. Soon after his admission to the hospital he became febrile. On January 15, 1943, a sample of his blood was sent to the Laboratory of Microbiology and Pathology of the Queensland Health Department and by means of guinea-pig inoculation a strain of Rickettsia was isolated. The strain was sent to F. M. Burnet, of the Walter and Eliza Hall Institute, Melbourne, in March, 1943, after undergoing two passages in guinea-pigs and three in mice, and later in the year, after one mishap, was sent to the United States Naval Medical Research Institute in America. Dissemination of the strain to other laboratories followed. Derrick and Brown point out that a feature of the Karp strain is that it was pathogenic for guinea-pigs from the first inoculation with human blood; only a minority of strains of *Rickettsia tsutsugamushi* are obviously pathogenic for guinea-pigs. Another unfortunate feature of the strain is the "distressing toll of laboratory infections" which has followed its dissemination. It was responsible for the death of a laboratory worker in Melbourne and laboratory infections have been reported from England and America. It has, however, been used extensively in experimental studies of scrub typhus and it is as well that the story of its original isolation and dissemination should be known.

[1] *The Lancet*, July 23, 1949.

Abstracts from Medical Literature.

THERAPEUTICS.

"Myanesin."

G. D. GAMMON AND J. A. CHURCHILL (*The American Journal of the Medical Sciences*, February, 1949) describe the effects of "Myanesin" upon the central nervous system. They state that "Myanesin" is one of a series of a substituted glycerol ethers. The drug is said to produce muscular relaxation, analgesia, ataxia and eventually paralysis. It is a local anæsthetic and also has a curare-like effect. It is said to abolish the tremors and rigidity of Parkinsonism. Given intravenously, one gramme of a 1% or 2% solution establishes the action of the drug for a few minutes up to an hour. To maintain the effect one milligramme per kilogram of body weight per minute must be infused. The maximum dose given was 87 milligrammes per kilogram. Tremors, athetosis, rigidity, spasticity, and the spasm of tetanus can be relieved. The effects are transitory. Oral medication with two or three gramme doses relieves Parkinsonian tremor to a degree for thirty to sixty minutes. Three grammes given twice daily for a month continue to be effective.

Pneumococcal Meningitis.

H. F. DOWLING, L. K. SWEET, J. A. ROBINSON, W. W. ZELLARS AND H. L. HIRSH (*The American Journal of the Medical Sciences*, February, 1949) describe the treatment of pneumococcal meningitis with massive doses of penicillin. Of 40 patients treated with sulphonamides alone, 93% died; only 62% died among 66 patients who received sulphonamides and penicillin. In a series of 21 cases the authors gave one million units of penicillin every two hours by the intramuscular route. The temperature fell below 101° F. by the seventh day. The spinal fluid concentration of penicillin was 0·08 to 1·25 units per millilitre within eight hours of the start of treatment. The blood concentration was 2·5 to 20 units per millilitre two hours after each injection. In only three cases was intrathecal penicillin therapy used for urgent reasons, 20,000 to 25,000 units being injected in one dose only. Continued intrathecal injections, which are generally advised for pneumococcal meningitis, do not give better results, and often cause serious complications. The authors recommend their method without the use of sulphonamides.

Anticoagulant Therapy.

W. T. FOLEY AND I. S. WRIGHT (*The American Journal of the Medical Sciences*, February, 1949) discuss long-term anticoagulant therapy for cardiovascular disease. They describe a series of patients suffering from auricular fibrillation, *phlebitis migrans*, recurrent thrombophlebitis and recurrent coronary thrombosis [*sic*] and myocardial infarction. The patients described suffered from a decided tendency to thrombosis or embolism. Treatment with heparin and dicoumarol was commenced in these patients after they had suffered one or more severe embolic phenomena. The authors make the suggestion that dicoumarol assisted in maintaining cardiac compensation. They admit the inconvenience of daily prothrombin tests and state that when a patient leaves hospital the prothrombin times can be estimated twice a week. These patients are those discharged from hospital on a regular dose of anticoagulant varying from 175 to 800 milligrammes per week. The aim is to maintain the prothrombin time at from thirty to thirty-five seconds. Variations occur at menstrual periods, and with variations in diet and alcohol intake. Nineteen cases are described. The dangers of the treatment are emphasized. None were observed in this series. Nevertheless sudden changes in prothrombin time were noted during treatment; these were controlled by large doses (70 milligrammes) of vitamin *K* given intravenously and whole blood transfusions. Minor hæmorrhages and skin ecchymoses were checked by these means. The authors emphasize the need of constant supervision of the prothrombin time when dicoumarol is being used. A check every seven to fourteen days is suggested to determine the prothrombin time.

Isotopes.

P. C. AEBERSOLD (*The Journal of the American Medical Association*, December 25, 1949) describes some of the uses to which radioactive isotopes are being put since they were made available outside the atomic energy project. A great variety of isotopes has been used experimentally in medicine, mainly P^{32}, radioactive phosphorus, and I^{131}, radioactive iodine. These isotopes have a half-life of only fourteen and eight days respectively. Gold, calcium, strontium and cobalt isotopes have also been used. Iodine, phosphorus and carbon isotopes have been used in physiology. Sulphur, sodium, potassium, iron and calcium isotopes have been employed in lesser degree. I^{131} has been used in the diagnosis and treatment of thyreoid disease and in the localization of brain tumours. P^{32} has been used in the study of leuchæmia, lymphosarcoma, polycythæmia and multiple myeloma. These are the more common uses in medical circles.

Œstrogen.

R. A. KIMBROUGH AND S. L. ISRAEL (*The Journal of the American Medical Association*, December 25, 1948) discuss the use and abuse of œstrogen. For post-menopausal vaginitis they advise the administration of 0·5 milligramme of diethylstilbœstrol or similar product daily by mouth for several weeks, discontinued after one month and repeated only for recrudescence of symptoms. For secondary amenorrhœa the main necessity is to ascertain the cause. If, however, uterine hypoplasia is the sole cause of amenorrhœa, treatment with œstrogen and the *corpus luteum* hormone progestin may help. Intramuscular injection of 2·5 milligrammes of œstradiol benzoate and 12·5 milligrammes of progesterone on two successive days, repeated for three consecutive months, may be effective. Oral administration of two milligrammes of diethylstilbœstrol daily for fifteen days, followed by sixty milligrammes of pregneninoline, provides a suitable schedule of oral therapy. The oral progestin therapy may be given for five days. Permanent results will not be obtained, however, by these means alone. Puerperal engorgement of the breasts may be relieved by one milligramme of diethylstilbœstrol given orally thrice daily for a week. For menopausal symptoms, flushes, sweats and nervous instability, when requiring specific treatment, 0·5 milligramme of diethylstilbœstrol given daily for a limited period gives good results, but it must be borne in mind that this treatment may induce bleeding, particularly post-menopausal bleeding, and simulate cancer of the uterus. Prostatic carcinoma and metastases thereof benefit by the giving of one or two milligrammes of diethylstilbœstrol thrice daily or five milligrammes of œstradiol benzoate or dipropionate by injection twice weekly. Irregular menstruation alone should not be treated by œstrogen because of the confusion which may be caused.

Combined Treatment of Paresis.

V. P. MAHONEY AND S. HAMMERMAN (*The Journal of Nervous and Mental Disease*, February, 1949) describe the results of combined treatment of general paralysis by penicillin, fever induced with typhoid organisms and electroshock. Penicillin 4·8 million units was given before and after seventy hours of fever therapy induced by injection of one and a half billion organisms per millilitre (8·5 to 30 millilitres of vaccine). Up to five hours of fever could be induced in one session. Behaviour difficulties were countered by electroshock, which is regarded as life saving. Of 44 patients with paresis, four died under treatment, 38 showed a pronounced degree of improvement, and 25 were discharged to out-patient status.

Ulcerative Colitis.

S. A. PORTIS (*The Journal of the American Medical Association*, January 22, 1949) discusses the ætiology and treatment of idiopathic ulcerative colitis. He considers that the parasympathetic nerves, vagus and sacropelvic, cause increased motility of the ileum and colon. The lesion at first is limited to the part of the colon innervated by the sacro-pelvic nerves. Owing to deficient action of pancreatic ferments digestion is not normal, and mucus which is normally present protecting the mucosa is destroyed by an excess of lysozyme, which is normally present in the intestine. In ulcerative colitis the lysozyme titre was increased from a normal 2·7 units to 56 units. The excessive lysozyme has a nervous origin, and the mucus being destroyed, tryptic digestion of the mucosa and invasion by bacteria may occur. The author then discusses the child's possessive interest in its excrement, and the money value placed on regular child defæcation by parents, and he describes two cases in which the demand for the return of a loan brought on ulcerative colitis. In this way the nervous factor is stressed. However, the author considers that the condition is medical and should not be treated by a psychiatrist. He recommends the usual type of non-residue diet, with a high content of protein, carbohydrates, vitamins and iron. Frequent feeds are advised. Smoking is prohibited, as prolonging hypoglycæmia, which is said to occur in ulcerative colitis. Atropine sulphate is given in doses from one two-hundredth to one-fiftieth of a grain after food and at bedtime, with phenobarbital one-quarter to three-quarters of a grain thrice daily. Sodium alkyl or lauryl sulphate 100 to 200 milli-

grammes thrice daily in enteric coated pills to bind lysozyme is advised. Phthalylsulphathiazole one or two grammes thrice daily is used to destroy bacteria. Crude liver extract two millilitres three times a week is also suggested.

The Anticonvulsive Action of Paraldehyde.

F. J. DE ELIO, P. G. DE JALON AND S. OBRADOR (*Journal of Neurology, Neurosurgery and Psychiatry*, February, 1949) discuss the anticonvulsive action of paraldehyde. The observations were based on clinical work as well as on experiments with cats. They show that paraldehyde is a substance of very little toxicity which can be injected intravenously in doses of up to 0·2 millilitre per kilogram without any ill effect. The depressing effect of the drug is greatest in the cerebral cortex, but it also occurs at the peripheral neuro-muscular junction and the ganglion synapses. Experimentally paraldehyde protects against the convulsions produced with strychnine or "Cardiazol". The authors recommend paraldehyde given intravenously in doses of 0·05 to 0·1 millilitre per kilogram as the best agent to break or stop convulsive discharges in clinical practice.

NEUROLOGY AND PSYCHIATRY.

Atypical Acoustic Nerve Tumours.

B. J. ALPERS AND F. M. FORSTER (*The Journal of Nervous and Mental Disease*, October, 1948) point out that cerebello-pontine angle tumours are sometimes regarded as falling into a universally typical pattern of tinnitus, progressive loss of hearing associated with headache, and staggering. Analysis, however, shows that vertigo may occur before tinnitus and loss of hearing. In one case the patient had a normal audiogram after twenty-eight months of symptoms. Loss of cochlear function may be slight.

Bulbar Poliomyelitis.

JOE R. BROWN AND A. B. BAKER (*The Journal of Nervous and Mental Disease*, January, 1949) state that whilst the death rate is high in bulbar poliomyelitis, the reasons are obscure. They claim that recent advances in neuro-anatomy and physiology allow us to analyse the underlying mechanisms. Their work is based on 59 cases of bulbar poliomyelitis among patients admitted to the University of Minnesota hospitals in a recent epidemic. The death rate was 44%. Four syndromes are described: (i) the cranial nerve nuclei group with lesions involving chiefly the motor nuclei of the cranial nerves; (ii) the respiratory centre group with the predominant lesions in the ventral reticular substance of the medulla; (iii) the circulatory centre group with lesions in the dorsal reticular substance; (iv) the encephalitic group with more diffuse lesions. In addition there is a fifth group in which the above are combined with paralysis of the peripheral respiratory mechanism. Most deaths occur as a result of involvement of respiratory or circulatory centres. In some cases respiratory obstruction is important. The following guides to management of the condition are suggested: (i) early elective tracheotomy; (ii) adequate suction of the pharynx and tracheotomy; (iii) oxygen mixtures with up to 40% of oxygen under positive pressure if pulmonary œdema is expected; (iv) adequate humidification of the gas mixture; (v) a careful checking of the oxygenation of the patient by means of arterial oxygen determinations and the use of the oximeter; (vi) frequent moving of the patient to avoid hypostasis and thrombophlebitis; (vii) maintenance of nutrition by nasal feeding; (viii) the use of penicillin prophylactically; (ix) the avoidance of sedative drugs and the use of stimulants; (x) early closure of the tracheotomy; (xi) unceasing watchfulness on the part of physician and special nurses, including the realization that mechanical devices are far from foolproof.

Basis of Schizophrenia.

J. M. NIELSEN (*The Journal of Nervous and Mental Disease*, April, 1948) states that when adults in the third or fourth decade of life suddenly develop a paranoid schizophrenic syndrome it is highly improbable that it is on a perfectly clear background. He finds that they have had the basis of schizophrenia for years previously. In some it is betrayed by a paranoid personality. Females have subnormal menstrual function. Males show persistent juvenility in sexual or general behaviour. All groups of psychotic *dementia præcox* show intellectual-emotional dissociation, a true splitting of the personality.

Syncope and Seizures.

JOHN KERSHMAN (*Journal of Neurology, Neurosurgery and Psychiatry*, February, 1949) discusses syncope and seizures. He describes the results of the electroencephalographic investigation of 114 patients who had episodes of transient dizziness, blurring of vision, blackout, abdominal sensations and weakness with or without loss of consciousness, but not associated with any definite convulsion. In all of them, between spells, the electroencephalogram showed a dysrhythmia. The symptoms were much the same as those which Gower described as vasovagal attacks. The author suggests that such disturbances are more epileptic in character than vascular in origin and have an origin probably in the hypothalamus.

Insanity Plea in Crime.

C. A. RYMER (*The American Journal of Psychiatry*, February, 1949) states that the criminal code still uses the McNaghten rules, which are concerned only with establishing whether the person accused knows the difference between right and wrong, or knowing the difference can refrain from doing the wrong. Legal procedure today leaves no other choice than that a man must be held absolutely guilty or absolutely innocent. A sharp line of demarcation cannot be drawn scientifically between sanity and insanity. Civil law does not attempt to require the patient to know the difference between right or wrong, but rather inquires into the patient's mental health and provides the necessary treatment. The author states that motives for behaviour, unconscious drives, aggression, love, hate and need for punishment, may well be included in determining a person's culpability. He reviews 750 cases, 129 for murder, in which a plea of insanity was made by the defendant; 75% of the subjects were examined by himself at the State Mental Hospital over a period of twenty years. One-third of the alleged murderers were considered insane. The non-psychotic subjects talked freely, claimed partial amnesia for the details of the crime, and all sincerely felt they must have been insane at the time of the crime, yet each felt that the plea of insanity as a defence to crime was abused.

Neurological Syndromes in Typhus Fever.

E. HERMAN (*The Journal of Nervous and Mental Disease*, January, 1949) draws attention to the 80,000 cases of typhus in Warsaw during 1941 and 1942. His department carried out examination and treatment of several thousand. Their neurological syndromes were extremely varied, including meningeal, ventricular, cephalic and encephalitic types. Extrapyramidal syndromes were frequent. Frontal syndromes produced the grasping reflex, in others cerebellar syndromes dominated the scene. It would seem that panencephalitis can attack any part of the nervous structure. In this series it is apparent that exogenetic factors, such as starvation, played an important part. The neurological and mental manifestations tend to occur with the exanthema, chiefly between the seventh and twelfth days of illness.

The Diagnosis of Epilepsy.

C. CURE, T. RASMUSSEN AND H. JASPER (*Archives of Neurology and Psychiatry*, June, 1948) discuss the use of intravenous injections of "Metrazol" or "Cardiazol" in the diagnosis of epilepsy. They state that it is a method that can be used instead of hyperventilation. Injections of these substances in small doses may produce an epileptic type of pattern in the electroencephalogram in susceptible persons. The threshold dosage in epileptic patients has been found to be much lower than in non-epileptic persons.

Psychomotor Epilepsy.

E. L. GIBBS, F. A. GIBBS AND B. FUSTER (*Archives of Neurology and Psychiatry*, October, 1948) recognize three chief epileptic syndromes: major, minor, and psychomotor seizures. They state that the last-mentioned group are rarely to be found in children, but are common in adults and are not as a rule controlled by medication; they can be diagnosed on clinical grounds alone. The chief clinical features of the psychomotor seizure are some degree of mental confusion without loss of consciousness, and movements which are simple, repetitive, automatic and purposeful, but poorly integrated. Manifestations of fear and rage, and shouting may be present. Hypersomnolence is frequent and some personality disorder is evident between seizures. In this study 300 patients with epilepsy whose electroencephalogram showed a psychomotor type of discharge had a history of psychomotor seizures, and in all cases foci could be demonstrated in the anterior temporal area on one or other or both sides. It is suggested that many of the personality disorders that are associated with the so-called epileptic personality are in fact psychomotor seizures.

Special Article.

FLUORESCENT LAMPS: A WARNING.

THE use of fluorescent lighting in factories, offices and shops has become increasingly widespread and this form of illumination is also being installed in some private homes. Because of reports from overseas it seems appropriate that a warning of the risks involved in the manufacture and careless handling of fluorescent tubes should be given.

In order to confer luminescent properties upon the lamps they are coated internally with a special mixture of "phosphor", which contains, *inter alia*, zinc oxide, beryllium oxide and silica. These substances, before being mixed with other powders and solvents, have been calcined together, the resulting material being known as zinc-beryllium-silicate. Beryllium "phosphors" are also used in neon signs and cathode ray tubes.

The industrial medical literature of the last two or three years, especially from the United States of America, but also from Britain, contains a number of articles describing serious, and sometimes fatal, damage to the lungs from the inhalation of dust during the manufacture of fluorescent tubes, and in other industries where beryllium compounds are made or used. Other reports have described skin lesions in persons who had cut themselves with glass from broken tubes. These pulmonary and skin effects have been attributed to beryllium (glucinum), an element which is chemically akin to calcium and magnesium and which occurs mainly in nature as beryl, a double silicate of aluminium and beryllium.

Two factories in New South Wales known to be manufacturing fluorescent tubes have been inspected by officers of the New South Wales Division of Industrial Hygiene. Both factories use powders containing beryllium. The managements were advised of the hazards involved, of the preventive measures necessary, and of the need for regular periodical medical examinations of the exposed employees, such examinations to include radiography of the chest. Seven employees from one factory were examined by the staff of this division, but no lesion attributable to beryllium was found.

I have no knowledge of any individual in New South Wales having suffered pulmonary or skin disease as a result of exposure to or contact with beryllium compounds. Cases, however, could have occurred of which I am unaware. Such cases could also have occurred without the attending medical practitioner's knowing or suspecting that beryllium might be an ætiological factor. In the absence, therefore, of any local first-hand knowledge as to the pathological effects believed to be due to beryllium, I think it is desirable that the following information, which is an extract from the *Monthly Review* of the Division of Industrial Hygiene and Safety Standards, New York State Department of Labour, April, 1949, should be brought under the notice of the medical profession in this country.

Pathological Manifestations.

Toxic responses to beryllium occur primarily in the lungs, upper respiratory tract and skin.

Respiratory Lesions.

Two types of respiratory disease have been described. One is an acute inflammatory reaction, varying from a mild pharyngitis or bronchitis to a massive pulmonary edema. It occurs principally among workers exposed to the soluble salts in the beryllium refining industry, and usually within a few days of a heavy exposure. Complete recovery is the rule, except for an occasional death from pulmonary edema.

The other respiratory form is a chronic pulmonary granulomatosis, developing after several months to years delay. It occurs primarily among workers handling beryllium phosphors, but has also been seen in those exposed to dusts or fumes of the metal and oxide. Cases have been reported among individuals living in the neighbourhood of beryllium processing plants. It is characterized by a nodular fibrosis giving a characteristic X-ray picture, and a diffuse thickening of the alveolar walls resulting in increased resistance to gaseous exchange across this membrane. The outstanding symptoms are fatigue, loss of appetite, weight loss out of proportion to the diminished food intake, and severe intractable cough. Treatment is limited to symptomatic relief, and a large percentage of those surviving remain at least partially incapacitated.

Cases are now being reported where the chronic form has apparently developed, without further exposure, several years after recovery from the acute. Conversely, it has occasionally been possible to obtain a history of an illness closely resembling the acute form and antedating the chronic by several months or years. Recently, a subacute type combining features of both the acute and chronic, with an intermediate latent period and duration, has been reported.

Skin Lesions.

In addition to the respiratory disease, several cutaneous reactions have been reported. In the beryllium refining industry, cases of acute dermatosis have occurred from contact with the soluble salts. Crystals embedded under the skin have produced foreign body reactions with ulceration. Extensively proliferating granulomata necessitating wide excision have developed following cuts from glass fragments in which particles of beryllium phosphor have remained. Subcutaneous granulomata have also been observed as a "metastatic" manifestation of advanced pulmonary disease.

In summarizing the pathological aspects of the pulmonary conditions seen in beryllium workers, Vorwald, of the Saranac Laboratory, New York, states as follows:

> Two distinct types of change are recognized: (1) the acute form, which resembles a pneumonic process, attended by edema, hemorrhage and exudate, but without the polymorphonuclear leukocytic response that accompanies an acute infectious process, and (2) the chronic form, which is characterized by the presence of a diffuse pulmonary reaction and scattered granulomatous lesions throughout both lungs.

According to Wilson, the lung changes associated with beryllium exposure have been most commonly confused radiologically with pulmonary sarcoidosis, miliary tuberculosis and silicosis, but other pneumonokonioses and other diseases may require differentiation.

With regard to the skin lesions, Grier, Nash and Freiman describe three cases of subcutaneous granuloma arising in persons who had cut themselves on broken fluorescent lamps coated with beryllium-containing phosphor. The authors state:

> The pathology of these lesions is similar to that seen in the skin lesions of two beryllium workers who had pulmonary granulomatosis, and essentially similar to the lung lesions seen in this disease. Because of the danger of recurrence, treatment should involve complete excision. If other cases of subcutaneous granuloma arising in this way are to be avoided, caution must be exercised in the disposal and salvage of burnt-out fluorescent lamps.

(In one of their cases the patient was a boy who was playing with friends, one of whom used an old tube as a baseball bat and hit a bottle with it!)

So that proper care may be taken to avoid the inhalation of dust or injury to the skin by broken glass, the safe disposal of used lamps is a matter of some importance and should be the responsibility of an individual who is fully informed as to the risks involved.

For the disposal of the tubes in industry, the Division of Industrial Hygiene and Safety Standards of the New York State Department of Labour in the *Monthly Review*, April, 1949, describes a method of breaking them under water with no danger from dust or flying glass. The Medical Advisory Committee on Beryllium recommends a method which involves breaking the lamps out of doors in a waste disposal area or in a ventilated hood, the breakage preferably being done within a waste container, the operator wearing an approved respirator. If the lamps are to be broken indoors, the work should be carried out in an isolated room and in a hood supplied with sufficient exhaust ventilation to provide an air intake of at least 125 linear feet per minute at all hood openings.

Broken lamps should not be placed in incinerators or dust bins, but should be thrown into either water or a rubbish dump, so that further human contact with the fragments is unlikely.

In a recent personal communication, the Director of the Division of Industrial Hygiene, New York State Department of Labour, advises that the general practice has been to mix the broken glass, preferably wet, with ashes or other inorganic refuse, and dispose of it in dumps or landfill. He states that the New York City Department of Sanitation has been depositing the intact tubes in landfill operations, crushing them with a bulldozer and then pushing the earth over them.

If lamps are broken accidentally in shops or offices or in the home, the glass fragments should be wetted, if possible, and collected with a shovel or by other means, so as to avoid actual handling of the fragments, and should be placed in a closed container for disposal as indicated above. It is suggested that consideration might be given to an arrangement whereby manufacturers and suppliers of fluorescent tubes, when replacing burnt-out intact lamps, would remove the latter for destruction at specially selected locations.

Recent advice to this division from both great Britain and the United States of America is that the use of beryllium in the manufacture of fluorescent lamps in those countries has been discontinued. However, in case beryllium lamps are being imported into this country and whilst beryllium "phosphors" are still being used locally, precautions are necessary in the manufacture, handling and disposal of these lighting tubes.

In conclusion, therefore, whilst, so far as is known, there is no danger from fluorescent lamps when intact, I would suggest that medical practitioners think of beryllium if the occupation of a patient with an unusual lung or skin condition is connected with the fluorescent lamp industry. In the case of a skin wound from a broken tube, the possibility that the glass has been coated with beryllium "phosphor" should be considered, so that a thorough search for glass fragments in the wound might be made. The staff of this division will be pleased to discuss these hazards more fully with any medical practitioner desiring further information.

GORDON C. SMITH.

Division of Industrial Hygiene,
Department of Public Health,
93 Macquarie Street,
Sydney.

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Public Health.

A SYMPOSIUM ON FEEDING PROBLEMS OF THE TODDLER.

A CONFERENCE on child health problems was held at the Australian Institute of Anatomy, Canberra, on July 17 and 18, 1948.[1] PROFESSOR J. C. SPENCE, who was in Australia as a visiting post-graduate lecturer, presided.

We are indebted to Dr. Eben H. Hipsley, of the Australian Institute of Anatomy, for supplying this report.

The subject "Feeding Problems of the Toddler" had been chosen for discussion, because the members of the conference were conscious of the frequency with which the complaint that her child "would not eat" was made by the mother; that anorexia varied from simply dawdling over meals to refusal of food and resentful hostility, or the so-called "negativistic behaviour". Meal times, instead of being events of relaxation and enjoyment, as should be the case, were apt to become battlefields where frustration and anger were the ruling passions. Both DR. V. L. COLLINS (Melbourne) and DR. KATE CAMPBELL (Melbourne) directed attention to the fact that the majority of toddlers who exhibited feeding problems with anorexia were in good nutritional state; only a few were poorly nourished. Both the well nourished and the poorly nourished groups deserved attention, the former because of the large numbers, and the latter because of the possibility of serious underlying disease.

Causes of Anorexia.

Physiological.

It was pointed out that the sensation of appetite was primarily due to the demand of the body tissues for nourishment; nutrients were needed for growth and repair of tissues, and to provide energy for various activities. If for any reason there was a lessening of that demand, loss of appetite would follow.

DR. V. L. COLLINS and DR. F. W. ARDEN (Brisbane) said that babies in the second year often did not require as much food as during the first year of life. For instance, Dr. Collins had found that at the Saint Gabriel's Baby Home in Melbourne, babies at the age of ten months consumed 10% more Calories than babies aged between one year and seven months and one year and nine months. That reduction of food intake in the second year of life seemed to be due to a slowing down in growth rate following the initial rapid spurt in the first year. Physical inactivity might sometimes have to be considered as a cause of anorexia. Anorexia at meal times might occur when the child was given ice-cream and lollies between meals. DR. R. A. J. STANTON (Perth) held the view that snacks, except perhaps fruit, should not be taken between meals.

Pathological.

It was appreciated that a poor appetite might be associated with organic conditions which must always be excluded. Dr. Kate Campbell discussed anorexia beginning in the second year, as distinct from the usual type of *anorexia nervosa* commencing in the early months of life. She analysed the case histories of 50 such private patients between the ages of one and two years, 19 males and 31 females. In 39 cases the primary cause was a fat intolerance, in seven cases the condition was due primarily to the stress of dentition, in three cases the primary cause was psychological, and in one case repeated upper respiratory tract infections were originally responsible. While those four conditions were the basic causes, it was found that the following exacerbating factors occurred: the nervous factor in 27 cases, teething as a secondary and not a primary factor in 15 cases, unhealthy tonsils in 13 cases, and upper respiratory tract infections as an ætiologically secondary factor in eight cases. In Dr. Campbell's series, dyspepsia was the primary cause in four-fifths of the cases, the nervous factor was involved either primarily or secondarily in three-fifths of the cases, and teething was the sole or exacerbating factor in two-fifths of the cases.

Psychological.

The participants at the conference unanimously agreed that not only was the majority of cases of anorexia in the second year of life of psychogenic origin, but that mismanagement by the mother was largely to blame. Even in the cases in which the children were poorly nourished, psychological factors were also important, as Dr. Campbell's analysis showed. The question to be answered was how the mother was at fault, and why.

Feeding Errors in Early Infancy.

It was freely agreed that the young baby needed food in the form of milk which he should be able to obtain by suckling his mother's breast when desired. In the past there had been a tendency for feeding schedules to be rigidly regulated by the clock, regardless of the demands of the infant. Recently C. A. Aldrich and E. S. Hewitt (*The Journal of the American Medical Association*, October 11, 1947) had reported their experience in feeding 100 infants up to their first birthday by the self-regulating method. They found that in general the usually prescribed schedules did not deviate to a great extent from the average intervals chosen by babies on a self-demand schedule. It was obvious, however, that whereas a rigidly prescribed routine of feeding could meet the requirements of the average baby, it could not possibly fit the needs of those whose natural rhythms deviated greatly from such averages. Both the precocious babies, who tended to lengthen the periods of their feeding times early in life, and the slower-to-change babies would be out of step. Since those deviations represented a large proportion of the total number of infants, the prescription of set schedules without regard to rhythms of individual infants must necessarily give rise to conflicts in the matter of feeding.

When the baby was forced to submit to practices arbitrarily determined by physicians, nurses and mothers, his relationship with his mother and the strong sense of security which he should have in her presence might be affected adversely. Anorexia, vomiting, excessive crying, breath-holding, and undue suckling were the common results. The physician who was unaware of the psychogenesis of those symptoms too frequently blamed the milk, and usually modified the food in consistency, content or volume, without looking beyond to such intangible factors as the mother-child relationship.

[1] The conference was held under the auspices of the trustees of the Anne MacKenzie and Charles MacKay Oration Funds.

The amount of the feed was often determined with little regard to the baby's appetite. Too often, when the baby fell asleep satisfied at the breast after a few minutes, he was stirred vigorously into wakefulness in order to make him take more food. Dr. V. L. Collins said that mothers should realize that feeding should be a pleasure for the baby, who should never be forced to take the breast. Placing the cot beside the mother's bed and encouraging feeding along self-demand principles would help to diminish some of the breast-feeding problems.

Feeding Errors in Late Infancy.

It was pointed out that Nature had provided the infant's first food, breast milk, but that she had given no indication, other than by appetite, when other foods should be commenced or when breast feeding should cease. In Australia it had been the custom to lay down arbitrary rules as to when additions should be made to the diets; a definite age for weaning was likewise advised. It was felt that no doubt those rules had been formulated as a result of vast experience and for that reason deserved respect, but that it should be realized that they were capable of wide variation. The institution of an "educational diet" had come to be widely accepted as a desirable practice for the infant when he reached the age of four to six months. Its purpose was to educate the infant in the taste and consistency of new foods and to teach him to chew and swallow. At first the new foods contributed little of nutritive value when compared with milk, which was the main food at that stage. Gradually, however, the introduced foods became increasingly important, and when the time came to wean the baby he would already be familiar with a wide range of flavours and consistencies.

At the age of three or four months the digestive system of the infant was well able to handle simple sugars, protein foods and starches. However, difficulty might be encountered in the digestion of excessive quantities of fat. In some clinics in the United States of America it had been observed that even meat in a suitably homogenized form was easily digested by infants, who usually enjoyed the food. DR. EBEN HIPSLEY (Canberra) described how the New Guinea native mother premasticated starchy vegetables, such as taro, sweet potato or banana, and fed the result to the young infant, who appeared to relish the morsel. Later when the infant was able to grasp he was given a larger piece of cooked vegetable, which he could gnaw at and suck. No harm had been reported from the addition of either the meat or the starchy foods to the diets of infants. On the contrary the educational value of the procedure was obvious, and in the case of meat supplements, Ruth Leverton and George Clarke, writing in *The Journal of the American Medical Association* of March 30, 1948, had reported that babies receiving meat supplements seemed to be of better physical appearance and had more satisfactory red cell and hæmoglobin values than controls who were fed milk diets alone. The truth with regard to the introduction of supplementary foods in the diets of infants appeared to be that, provided fatty foods were avoided, there was considerable latitude in the choice of educational foods. The likes and dislikes shown by the infant towards certain foods could, within moderation, be respected.

Dr. V. L. Collins said that a common error in feeding, which might occur when educational feeding was commenced, was that the mother sometimes tried to force the baby to eat the foods she thought he must have, and that she would not wait until he was willing to take the food. The child then became antagonistic and would not eat.

It was felt that, although it was generally taught that the infant should be weaned at the age of about nine months, it should be realized that that age was arbitrary and considerable latitude was permissible. B. Spock, in a paper published in *The Journal of the American Medical Association* of March 30, 1948, was of the opinion that the child should not be weaned until he showed readiness, even though it was not until the middle of the second year. It was probable, however, that long before he reached that age he would want to show his independence of his mother and wean himself, particularly if he had been educated in the use of foods, hands, spoons and cups.

Errors in Feeding of Toddlers.

In a discussion on errors in feeding of toddlers it was agreed that the young child was able to digest the elements of an average balanced diet with the exception that fat might sometimes cause difficulty. By the time he was in his second year he had developed a fairly efficient dentition and it was surprising what use he could make of it if given the opportunity. It would be some time before the child would develop the fine skills necessary for using spoon, knife and fork, but he would be capable of feeding himself with his own fingers and drinking from a mug. During that stage of development, he would be learning rapidly by precept and example and would require guidance. Dr. R. A. J. Stanton expressed the opinion that the child should dine often with his parents, from whom he would be able to learn much.

Dr. Eben Hipsley said that from a physiological point of view there was little doubt that the right time to take food was when an appetite had been created for it by the demands of growth and activity. The correct sequence of the natural rhythm of living and of healthy physiological activities was work, food and rest. The main meal of the day should come as a climax to activity and towards the end of the day. DR. LORIMER DODS (Sydney) commented that the problem of anorexia was rarely encountered among the infants of those peaceful and phlegmatic labouring-class parents who followed the natural rhythm of living so aptly defined by Dr. Hipsley as "work, food and rest". He said that in his experience the problem of anorexia belonged in the main to a different type of family, the members of which lived "over-anxiously" and failed to achieve for themselves or their infants the peaceful rhythm of their more casual and confident neighbours.

MISS G. E. PENDRED (Melbourne) said that a special study of problems had been made at the Lady Gowrie Child Centres. In some cases it was found that the meals at home consisted of consistently too large servings, which defeated the child with a normal but small appetite. In others, the eating problem could be traced to the food's being badly prepared and cooked, for example, strings left on beans, hard cores in cooked fruit *et cetera*.

Another error, Miss Pendred said, was that sometimes foods difficult to manage were served and too much stress was laid too early on "table manners". Parents should be helped to prepare food in such ways that a young child could manage it easily for himself, for example, potatoes could be served in pieces so that the child could pick them up with his fingers. These points of ease of handling food were also emphasized by DR. NORMA KELSO (Melbourne) and Dr. R. A. J. Stanton.

Miss Pendred said that in the junior nurseries (two to three years) frequently it was found that a child prior to entering the nursery had been given only a *purée* of vegetables and other soft foods and had not had any practice in chewing or in recognizing and enjoying the different food flavours. When presented with food that required some effort by chewing those children were too lazy to make any effort. In cases in which children rejected some particular food, mothers were advised not to make an issue of it, but to accept the fact calmly and either quietly remove the food and serve it at a later meal without comment or better still ask the child just to taste it.

All agreed with DR. E. B. SIMS (Adelaide) and Dr. V. L. Collins that parents (and grandparents) too often followed some rigid nutritional doctrine. Very few foods were absolutely essential and could not be replaced by acceptable substitutes. A love of combat in the child might easily be aroused if unpopular or distasteful foods were persistently offered with entreaties, threats or ill-concealed parental emotion. Dr. Collins stressed the fact that mothers should not worry if the diet was not balanced each day, but should be satisfied with it balanced over a week or so.

Both Dr. Collins and Dr. Sims pointed out that a habit of food refusal often appeared to have been initiated quite early as a result of some accidental circumstance, such as a transient infection or teething. If the physiological anorexia associated with such an event was not appreciated by the parents, it was possible that persistence in efforts to feed a reluctant child would set up an aversion to food that would persist long after the original cause was past and forgotten.

The Origin of Wrong Attitudes of Mothers.

The question was then considered why the mother developed wrong attitudes. It was pointed out that the mother depended for her knowledge of mothercraft on the personal experiences of her own environment, on the instructions she received from doctors and nurses, on an assortment of knowledge obtained from reference books, newspaper articles and advertisements, and on conversations with her mother or neighbours. If she had ever lived in a household where young babies were being brought up, she would have ample opportunity to study infant care at close hand. At the present day when small families were common, less opportunity for learning infant care at home existed. It was the mothercraft schools which were responsible for most of the teaching of mothercraft, but ultimately it was the doctor who indoctrinated the mothercraft nurse.

DR. F. W. CLEMENTS (Canberra) said that doctors, nutritionists and dietitians were often to blame for setting rigid standards and for teaching that the child must have certain foods every day. They had inculcated those ideas in mothercraft nurses, who passed them on to the mothers, who believed them implicitly. There was no reason why the child must have certain specified foods every day or even at all. If the child ate a range of foods he would then obtain his nutritional requirements. The trouble was that too many people in authority had a rigid attitude toward food. They must reeducate the older nurses and teach new ones a more flexible outlook. With that DR. MARGARET HARPER (Sydney) agreed, and said that nurses were now being taught that diet rules were not rigid.

Conclusions.

There was apparent unanimity of opinion at the conference that feeding problems of psychological origin were common amongst toddlers. Those had been mostly due to the too rigid enforcement of arbitrary doctrines, first by the doctors and nutritionists, then by the mothercraft nurses, and lastly by the mothers themselves. A change from this rigid attitude to a more flexible approach would be beneficial. Feeding the infant along the self-demand principle, where it was compatible with the exigencies of modern life, had undoubtedly much to commend it. The infant could safely be allowed some latitude in the choice of his own diet, and at all events no force should be exercised.

Correspondence.

AN UNUSUAL HAPPENING DURING TONSILLECTOMY.

SIR: The following incident during a tonsillectomy operation seems worth recording owing to its unusualness and also on account of the position in which the missing tonsil was found.

During the operation under machine ether through a Boyle-Davis gag the left tonsil was dissected in the usual way. The snare was used to cut through the "stalk", and the tonsil disappeared as the snare was being withdrawn.

Owing to the fact that the child was well anæsthetized and without the power of swallowing, the tonsil could not have gone down the œsophagus, and as the larynx or rather the epiglottis (pharyngeal surface) was clearly visible, the tonsil was not there.

Gentle search by the finger failed to reveal it in the naso-pharynx. I decided to go on with the operation as I felt I knew where the tonsil was. I removed the other tonsil and then curetted the adenoids, but there was still no sign of it.

I then passed a Eustachian catheter through the right nostril with my left forefinger in the naso-pharynx and felt the tonsil being pushed out end on from the right choana.

I have been doing this work for twenty-seven or twenty-eight years and have never had this experience before. I think that one MEDICAL JOURNAL OF AUSTRALIA of the early twenties recorded a case from South Australia where the whole tonsil was inhaled into the lungs.

It seems impossible that fairly large tonsils can go into these openings and consequently it seems worth recording, as it may happen to somebody else and he may be a bit overwhelmed by the apparent successful conjuring trick he has performed.

Yours, etc.,
C. E. S. JACKSON.

Ballow Chambers,
Wickham Terrace,
Brisbane.
July 28, 1949.

CORONARY OCCLUSION.

SIR: I am grateful to Dr. Keith Bowden for his letter in your issue of July 9, 1949, in which he stated that among 317 fatal cases of coronary insufficiency examined by him, coronary thrombosis was observed in 18 cases only. This supports Wright Smith's observations in nearly 500 cases, that coronary thrombosis is a rare cause of coronary occlusion.

The correct diagnosis in acute coronary accidents, therefore, is not coronary "thrombosis" but coronary occlusion. It is curious that the diagnosis of coronary "thrombosis" has become widely accepted both in Britain and America. In the *British Medical Journal*, April 2, 1949, there is a leading article on "coronary thrombosis" treated with anti-coagulant drugs. In that article there are references to the investigations of the Committee of the American Heart Association concerning "coronary thrombosis". The emphasis is always on "thrombosis". One author has even suggested giving dicoumarol to patients with angina of effort to prevent them getting coronary "thrombosis"—a condition against which the chances are 10 to 1, according to Wright Smith and Bowden. In THE MEDICAL JOURNAL OF AUSTRALIA the acute coronary incidents are usually referred to as coronary insufficiency or coronary occlusion, and this would appear to be the best diagnosis from a clinical viewpoint.

Yours, etc.,
G. C. WILLCOCKS, F.R.C.P.

143 Macquarie Street,
Sydney,
July 28, 1949.

OPHTHALMOLOGY IN AMERICA.

SIR: The chief points British and Australian ophthalmologists can learn from their American colleagues are, I think:

1. Gloves should be worn by all doctors and nurses at eye operations.

2. Cataracts should be extracted by the intracapsular method.

3. Corneal grafts should replace optical iridectomies and tattooing for corneal opacities.

4. Aniseikonic clinics should be established at eye hospitals.

Yours, etc.,
F. SIMPSON, D.O. (Oxon.), M.B., B.S. (Adelaide).

105 Saint George's Terrace,
Perth,
Western Australia.
July 29, 1949.

DIGITALIS AND EXTRASYSTOLES.

SIR: In the review of the "Textbook of Medical Treatment" by Dunlop, Davidson and MacNee, published in this journal on July 2, 1949, appear the following lines: "We cannot enthuse, however, about the statement (on page 638 and 661) that 'digitalis is of occasional advantage in the abolition of extra systoles when these are troublesome to the patient'." To my mind the expression "one cannot enthuse" is indeed the right one. But all the less I would by no means miss digitalis in the treatment of extrasystoles. I have experienced its beneficial effect on my own extrasystoles for more than forty years. I never had any organic heart ailment, but I had to feel "troublesome" extrasystoles since my youth. They came sometimes very seldom, at other times, however, they occurred rather often and then lasted for weeks. The Wenckebach pills stopped them almost always promptly in four to seven days. Dutch-born Wenckebach, the once famous cardiologist of Vienna, constructed the pills against extrasystoles. The prescription, quoted from Scherf and Boyd's book, is as follows:

> *Quininis sulphurici grammata quatuor* 4·0.
> *Pulv. fol. Digitalis grammata duo* 2·0.
> *Strychnini nitrici centigrta sex* 0·06.
> *Massa pilularis ut fiant pilul. Centum.*
> Ds. 3-6 pills to be taken daily.

A similar combination without digitalis proved without effect on me. I had about the same experience on a large number of patients too; in a few cases the quinine-strychnine combination was efficient in itself.

Present opinions about the use of digitalis against extrasystoles are still of very wide discrepancy. Some authors do not mention it at all, some advocate it, others do not like or even contraindicate it, a few feel that further observations are still wanted. I think it is worth while to quote two interesting opinions here. In his famous book "Diseases of the Heart", Edens, one of the best experts on digitalis, writes the following:

> Against certain extrasystoles the most efficient is digitalis in small doses (5 centigrammes 1-3 times per diem). Digitalis provokes extrasystoles, but is also able to stop them. Digitalis has a Janus face and shows alternately the one or the other side.

A quotation from the quite recent book of Scherf and Boyd:

> It may seem strange that a drug which frequently produces extrasystoles should also be employed to

abolish them. The extrasystoles which are not the consequence of digitalis disappear during digitalis treatment. Often small doses, such as 0·2 gramme daily for four or five days, suffice for this purpose. It is, however, rarely necessary to give digitalis to abolish extrasystoles.

I completely agree with the last sentence. I do not "enthuse" either about the use of digitalis against extrasystoles, and would not recommend its use as a routine in every case. If, however, diet, rest, sedatives, quinine, quinidine *et cetera* do not help, let us undertake a trial with digitalis: it is safe.

Yours, etc.,

CHARLES ENGEL.

22 Ocean Avenue,
Edgecliff,
N.S.W.
August 5, 1949.

References.

Edens, Ernst (1929), *"Die Krankheiten des Herzens und der Gefässe"*, pages 483 and 524.
Scherf, David, and Boyd, Linn I. (1948), "Cardiovascular Diseases", page 565.

ACCIDENTAL AIR EMBOLISM IN A BLOOD DONOR.

SIR: The further mention of air embolism in blood donors by Dr. W. T. Agar in the journal of July 23 prompts me to direct attention to one or two points in the design and use of "Soluvac" apparatus which may help to eliminate this unpleasant mishap.

Firstly, in any kind of apparatus designed to apply a negative pressure when necessary, some relatively restricted air outlet must be a feature, and it is therefore important to ensure at the outset that this passage-way is clear. The most positive means is to use the suction apparatus to draw the anticoagulant solution from an ampoule into the flask (rather than to have it sterilized *in situ*, as is the practice of some operators). At least one of the cases earlier mentioned in the journal would have been avoided had this practice been followed.

Secondly, it may be of interest that the bung perforator needle in the "Soluvac" apparatus was made short deliberately, in order to prevent the type of occurrence reported by Dr. Agar. It is intended that its orifice remain sheltered within the recess on the underside of the bung. The use of a larger needle, as suggested by Dr. Melville Kelly, would tend to defeat this purpose.

Incidentally, the length of needle provided is not related to the length of the perforator, which will actually house a needle up to one and a quarter inches long.

The instructions for use of the set also provide that the large needle of the collecting tube be inserted "deeply" into the flask. If this is done the incoming blood is carried down past the bung, the under surface of which should not be soiled.

Yours, etc.,

C. WALLACE ROSS,
Technical Administrator, Medical Division, Drug Houses of Australia, Limited.

20-22 O'Connell Street,
Sydney,
August 1, 1949.

STREPTOMYCIN AND TUBERCULOSIS.

SIR: I would like to make some remarks about the present trend in the use of streptomycin for tuberculosis. The position of streptomycin in the concept of tuberculosis is beginning to define itself and it has been recommended by most authorities that it should be reserved only for certain types of the disease; concurrence of opinions on its use is at least in (i) tuberculous meningitis, (ii) tuberculosis of the respiratory passages and (iii) the miliary type of tuberculosis.

In the more chronic type of disease the use of streptomycin has been condemned, mainly on the grounds that it has little apparent effect on the lesions themselves and on the subsequent course of the disease.

Recently its use in the acute exudative type of tuberculosis has fallen into disfavour. The reasons postulated are that the drug should be withheld in case the patient should ever come to the position where he may require surgical procedures, when it can be used in the event of a complicating spread.

Streptomycin is usually withheld even as a routine cover to thoracoplasty for tuberculosis, although some surgeons apparently temporize with pneumonectomy, despite the theoretical fact that post-operative spread to the contralateral lung is more common in the former procedure. The basis of the reasons for this withholding of streptomycin appears to be the danger of the establishment of a drug-resistant strain in the patient and also the fact that doses may be cumulative in this regard.

The present position seems to be one urging for a more careful selection of suitable patients for streptomycin therapy and a narrowing down of this "suitable" field.

I would like to plead for the so-called "unsuitable" cases, who are denied this drug on the grounds previously mentioned. I maintain that chronic tuberculous patients with clinical exacerbations of the disease, especially in those despite bed rest, should not be penalized. These are all acutely sick tuberculous patients and they should have every treatment possible to relieve the present state of their disease. It is in these patients that benefit does occur, at least clinically, from the drug, and they are entitled to at least temporary relief.

Similarly, I think that if a tuberculous patient is to undergo any form of major surgery he should have every therapy in our power to tide him over a difficult passage and not to wait until further complications appear before combating them.

Again, streptomycin may benefit many tuberculous patients by improving previously inoperable cases sufficiently to allow collapse therapy or resection.

Naturally, surgeons would like to have each patient present themselves for surgery with a *"non est"* streptomycin chart, as they will then have something up their sleeve in a case of emergency, but this is rather a selfish outlook by which many borderline cases may never reach their hands.

A review of the literature shows that resistant strains to concentrations of streptomycin to be expected in the bloodstream in a daily dosage of one gramme, appear to become manifest between the fortieth to sixtieth day of therapy. No case has been shown developing resistant strains before the thirtieth day of therapy.

Acute clinical manifestations of tuberculosis usually respond to streptomycin treatment within a few days, and if the drug is used for symptomatic reasons not more than a few grammes' total dosage need be given. After all, streptomycin is not going to be the only drug ever to be effectively used against tuberculosis; other drugs are appearing which may overcome possible streptomycin-resistant tubercle bacilli.

Finally, I agree that a rational outlook on streptomycin be retained, but that a drug, which is the first to have been demonstrated to beneficially alter the course of tuberculosis, should have been described as "stupid", and is in danger of being discarded, is surely a crime against the today of tuberculous patients, despite the postulation of their hazardous morrow.

Yours, etc.,

R. MUNRO FORD.

Chest Clinic,
Royal Adelaide Hospital,
Adelaide.
August 9, 1949.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course of Lectures for the Primary F.R.A.C.S. Examination in the University of Sydney.

THE Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the New South Wales Committee of the Royal Australasian College of Surgeons, will conduct a course of lectures during October, 1949, suitable for candidates for the primary F.R.A.C.S. examination to be held in November next. Lectures will be held in the afternoons only and will occupy most of the month of October.

Application to attend should be made to the Course Secretary of the Committee, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483.

Seminar in Medical Statistics.

Arrangements have been made for a seminar in medical statistics to be held on Wednesday, September 28, at 5.15 p.m., in the Maitland Lecture Theatre, Sydney Hospital. The

subject will be "The Results of Some Follow-ups on Known Diabetics in New South Wales", and the speakers will be Dr. J. Kempson Maddox and Dr. John Cobley. Any workers in medicine or related sciences are welcome to attend.

Examinations.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the following candidates have passed in the recent examinations for the degree of Master of Surgery, Part II: R. D. Condon, A. F. Dwyer, D. de la F. Henry, E. V. Peipman, T. F. Rose, J. J. Smyth, A. L. Stephenson.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 56, of August 4, 1949.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Termination of Appointment.—The appointment of John Wallis Hornbrook as Surgeon Lieutenant is terminated, dated 13th May, 1949.

Royal Australian Naval Volunteer Reserve.

Appointment.—Stephen Creighton Suggit is appointed Surgeon Commander, with seniority in rank of 31st December, 1946, dated 11th March, 1948.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps.

VX504193 Captain F. Grunseit is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (2nd Military District), 24th March, 1949 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 30 of 1949, promulgated in *Commonwealth Gazette* No. 21 of 1949).

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical): To be Captain (Provisionally), 25th May, 1949.—1/25060 Ronald Thomas Todd.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/66219 Major D. R. Reid is appointed from the Reserve of Officers with regimental seniority in accordance with Army seniority and is borne supernumerary to the authorized establishment of Majors, with pay and allowances of Captain (at his own request), 1st May, 1948. To be Captain (provisionally), 26th April, 1949.—2/107771 Ronald James Jennaway.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/50065 Lieutenant-Colonel J. S. Peters is appointed from the Reserve of Officers with regimental seniority in accordance with Army seniority, and is borne supernumerary to the authorized establishment of Lieutenant-Colonels with pay and allowances of Major (at his own request), 1st April, 1948. To be Temporary Colonel, 26th May, 1949.—3/51002 Lieutenant-Colonel (Honorary Colonel) W. W. Lempriere, D.S.O.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—5/32053 Lieutenant-Colonel (Honorary Colonel) F. K. Wallace, O.B.E., is absorbed within a vacancy in the authorized establishment of Lieutenant-Colonels, receives pay and allowances of that rank and is appointed to command 7th/13th Field Ambulance, 1st July, 1949. 5/15975 Honorary Captain D. C. Foster is appointed from the Reserve of Officers and to be Captain (provisionally), 2nd May, 1949.

DISEASES NOTIFIABLE IN EACH STATE AND TERRITORY OF AUSTRALIA AND NOTIFICATIONS FOR THE WEEK ENDED JULY 30, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	9(8)	..	..	*	..	..	..	9
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	3(2)	2(1)	..	2	..	..	..	..	7
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	3(2)	..	..	..	..	..	3
Diphtheria	5(3)	3(2)	4(1)	..	3(2)	1	..	..	16
Dysentery(*b*)	*	4(4)	..	..	..	..	..	..	4
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	2(2)	*	*	..	..	2
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	1(1)	*	*	..	*	1
Leprosy	..	..	..	..	1	..	..	..	1
Malaria(*c*)	*	(*c*)	1(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	1(*c*)
Measles	*	*	*	118(31)	*	*	..	..	118
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	12(11)	1(1)	10(10)	2(2)	..	..	..	25
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	1	..	1(1)	1(1)	..	..	..	..	3
Rubella	*	*	*	*	*	*	..	..	..
Scarlet Fever	31(21)	21(11)	..	9(7)	5(5)	3(1)	..	..	69
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	2(1)	*	*	*	*	*	2
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	34(16)	17(6)	11(3)	9(3)	4(4)	2(1)	..	1	78
Typhoid Fever(*e*)	..	..	..	..	..	..	..	..	..
Typhus (Endemic)(*f*)	..	..	1	..	..	..	..	..	1
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	3	*	*	*	*	*	3
Whooping Cough	*	*	*	34(7)	*	*	..	..	34
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944-1945. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Northern Territory.
* Not notifiable.
(*a*) Includes "Mossman" and "Sarina" fevers. (*b*) Includes amœbic and bacillary. (*c*) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (*d*) Includes all forms except in New South Wales and Northern Territory, where only pulmonary tuberculosis is notifiable. (*e*) Includes enteric fever and paratyphoid fevers. (*f*) Cases reported are all of the mild type known as Brill's disease or endemic typhus (including scrub and urban types). (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

5th Military District.—The following officers are placed upon the Retired List (5th Military District) with permission to retain their rank and wear the prescribed uniform, 28th March, 1949: Lieutenant-Colonels L. A. Hayward and C. H. Leedman, M.C., Major H. G. D. Breidahl and Honorary Majors F. T. Beamish, O.B.E., and H. B. Gill. The following officers are retired, 28th March, 1949: Honorary Captains R. H. N. Connell and W. P. Yates.

Obituary.

HAROLD SKIPTON STACY.

THE following tribute to the late Dr. Harold Skipton Stacy has been received from Dr. H. Roger Cope.

I would like to pay a last tribute to Harold Skipton Stacy, M.D. He was one of my oldest friends. We entered the University Medical School together in 1892 and from then onwards have been close friends. He was a man who I know would not anticipate any post-mortem eulogies. He had his share of the faults which most of us possess, but they were so screened by his high qualities, his gay laughter, his courage and loyalty that they were forgotten and hidden in the silent mind. His courage during his late illness was remarkable. Often he said to me: "Why should I grumble when I think of what Nell suffered for nearly two years?" And Nell was his wife whom he loved. He laughed at money and the want of it. He laughed at his complaints. It was sad to see what was once a straight athletic figure come down to see me at Cronulla. I met him at the train, an old man bent in years but still laughing. He took a long time to get in the car and yet a longer time to get out, and yet he was still sitting on medical boards to help things along. When at the last rites at the crematorium someone made contact and the casket moved in slow silence to its destination, I thought of our long friendship to be tried by the furnace and delivered as gold. May his spirit rest in peace. *Vale*, Harold!

LESLIE FRANCIS CLAREMONT.

WE regret to announce the death of Dr. Leslie Francis Claremont, which occurred on August 9, 1949, at Lindfield, New South Wales.

Notice.

A COURSE of six lectures on "Psychiatry and the Ex-Serviceman" has been arranged by the New South Wales Branch of the Repatriation Department. They will be delivered by Dr. Alan Stoller, specialist in psychological medicine, in the medical library, Grace Building, corner of York and King Streets, Sydney, at 5 p.m. on the following dates: September 2, 1949, "Present-Day Psychiatric Concepts"; September 5, 1949, "Causation of Mental Disorders"; September 7, 1949, "Classification of Mental Disorders"; September 9, 1949, "Psychiatry and General Medicine"; September 12, 1949, "Modern Trends in Psychiatric Therapy"; September 14, 1949, "Psychotherapy in General Medical Practice". All local medical officers of the Repatriation Department and all students of psychiatry are invited to be present.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Tarn, Joseph Henry Teesdale, M.B., B.S., 1947 (Univ. Sydney), 4 Clifford Street, Gordon.

Farb, Max, M.B., Ch.B., 1945 (Univ. New Zealand), 12 Ormonde Parade, Hurstville.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Ey, Geoffrey Thomas, M.B., B.S., 1947 (Univ. Adelaide), 3 Wheaton Road, St. Peters, South Australia.

Pavy, Rosemary Dorothea, M.B., B.S., 1948 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Cherry, Aileen Percival, M.B., B.S., 1949 (Univ. Adelaide), 23 Brougham Place, Alberton, South Australia.

Diary for the Month.

AUG. 23.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 24.—Victorian Branch, B.M.A.: Council Meeting.
AUG. 25.—New South Wales Branch, B.M.A.: Branch Meeting.
AUG. 25.—South Australian Branch, B.M.A.: Branch Meeting.
AUG. 26.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 1.—New South Wales Branch, B.M.A.: Special Groups Committee.
SEPT. 1.—South Australian Branch, B.M.A.: Council Meeting.
SEPT. 2.—Queensland Branch, B.M.A.: Branch Meeting.
SEPT. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 7.—Victorian Branch, B.M.A.: Branch Meeting.
SEPT. 7.—Western Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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